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AN EXPISCATION OF ACUTE DELIRIUM.

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There is probably no department of medicine in which the scientific study of disease affords so little of mental satisfaction as in that which treats of insanity. The reason of this is not far to seek; as I have formerly insisted upon, the various insanities to which names have been given by authors, as though they were distinct diseases, are in great part nothing more than symptom-groups, one of these groups often containing two or more diseases, and one disease reappearing in two or more groups. As in the former time, physicians talked about dropsy as a disease, and now this alleged disease is resolved into heart, renal, and kidney affections, so at present we speak of manias and melancholias, which in the aftertime will, we hope, be resolved into various organic affections.

Our knowledge of cerebral pathology in its relation to cerebral action is so imperfect that it seems almost impossible for the alienist to get much firm ground under his feet; indeed, it still remains doubtful whether it will ever be possible to get a complete pathological basis for our study of insanity, unless, indeed, some great genius shall arise and invent new methods of investigation. The matter is made more difficult and the value of our present knowledge more problematic by the fact that we have no definite, positive proof at present that corresponding lesions produce in different individual brains the same symptoms; whilst we do know that different lesions will produce closely similar symptoms. Nevertheless, by splitting off here and there a group of cases whose pathology has been made out, we may hope little by little to obtain more and more definitude of knowledge, and continually reduce

in number and importance the symptomatic disease-groups of unknown pathology.

In chronic insanity such a long time usually elapses between the commencement of the process and the death of the individual that secondary brain-alteration must continually obscure any original structural deviations; pathological study in chronic insanities is therefore more hopeless than in the active disorders. It is plain that in an attempted elucidation of the essential nature of insanity very little value attaches to reports of cases in which recovery has occurred, and in which, therefore, no pathological study has been possible. The literature of medicine teems with the reports of such cases; indeed, there has been such an accumulation that it is hardly probable that our knowledge can be advanced by further records unless these records be directed to the elucidation of one or two clinical points which seem yet in doubt, as, for instance, the question of bodily temperature in acute mania.

Again, reports of fatal cases in which no exhaustive microscopic examination has been made are plainly of no more value than are those of cases in which there has not been a post-mortem. In fact, such reports are worse than useless, because they may well be misleading. Further, when it is remembered that it is possible for an insanity to be of toxic origin the importance of bacteriological and chemical studies of fatal acute insanities becomes manifest; but such studies are as yet very few.

It seems to me, also, a matter of great importance that a clear consensus of professional opinion should be reached as to what are the problems which are at this time next in order of discussion; or, in other words, what questions the alienist should now strive to answer.

There are commonly recognized a number of acute insanities which may for our purposes be separated into two groups: Group 1, acute melancholia, acute stupor; Group 2, acute mania, acute delirium (typho-mania or Luther Bell's mania), confusional insanity, *i. e.*, the mania which follows shock of injuries or surgical operations, febrile disease or certain chronic poisonings, such as alcohol, cocaine, morphine, etc., intoxications mania due to direct action of poisons.

The questions which seem to me to be most pressing for answer on the part of the alienist are: First, what is the relation between acute melancholia and primary dementia? Second, what is the difference between acute mania and acute delirium or typho-mania? Third, do the different manias which follow acute diseases rest upon one pathological basis, or are they of diverse nature; and if this be so, is it possible at this time to cleave off from the block one or more distinct diseases?

In the present article I do not propose to discuss the relations between acute melancholia and primary dementia, but shall confine my attention to what we may term the "manias," *i. e.*, Group 2. Among recent writers there is much difference of opinion as to the relations

between acute delirium and acute mania; some teaching that the two disorders are essentially distinct, others that they are simply different grades of the same disease.

A study of the nature of acute delirium does not so closely involve a study of the relations between these two disorders as would an investigation into the nature of an acute mania. The opportunities for early post-mortems are so much more frequent in acute delirium than in acute mania that there is much greater hope of elucidating its nature than there is of discovering that of acute mania, so that the immediately pressing question seems to me to be as to the nature of acute delirium. Nevertheless, I shall discuss briefly the clinical relations of the two affections.

The symptomatic differences between acute delirium and acute mania are found in the suddenness of the onset of acute delirium, in the rapidity of development of typhoid symptoms, and especially in the presence of a pronounced febrile reaction. In a typical case of acute mania there is, preceding the outbreak of symptoms of exaltation, usually a period of depression, and this period, it is true, is often, perhaps usually, wanting in cases of acute delirium; but it would seem certain that acute mania may develop without previous depression, and that an acute delirium may be preceded by melancholic symptoms; indeed, in the case hereinafter narrated the primary mental condition was one of depression, and the early delusions were depressive, being of persecution.

So far as concerns the mental symptoms the differences between the two diseases are therefore rather in regard to intensity than as to character. How is it with the bodily symptoms? Some writers affirm that in one disease the patient very rarely, if ever, dies, whilst in the other the natural course is toward an early death. Assuredly illogical, however, is it to base diagnostic difference on the occurrence or non-occurrence of death; and assuredly, also, cases which seem in the onset to be instances of the graver malady do occasionally recover. It would seem, therefore, that so far as the question of prognosis is concerned the difference between acute mania and acute delirium is difference in degree rather than in kind; and the question naturally arises, Is this true of all the physical symptoms so called, or, in other words, are these physical symptoms, like the mental symptoms, different simply in intensity and not in kind?

If there be any salient symptomatic difference in the two disorders, it would seem to be in regard to the bodily temperature. In acute mania the temperature is generally stated not to be distinctly altered; whilst in acute delirium the fever rises to a considerable height. Krafft-Ebing lays great stress upon this difference, affirming that the rise of temperature in any case of mania to 100.5° F. indicates very strongly the presence of delirium acutum. The severe cases of acute delirium are so sudden and rapid in their course that I think they are more frequent outside

than within asylums; whilst acute manias are so prolonged and so difficult of management that the great majority of cases pass into the hands of the asylum-alienists. In order to get, if possible, the experience of these practitioners upon the point in question, I have written to some of those whom I conceived to have had largest experience, and received in most instances courteous, but often not very satisfactory, replies.

Dr. Morgan Cloud, first assistant physician in the Kansas State Insane Asylum, writes: "In cases of acute mania admitted here the temperature ranges from 99.5° F. to 100.5° F. in the majority of cases; in a few cases the temperature rises as high as 103° F. for a short time only, and I am inclined to think that in these cases there is some other cause than acute mania for the extreme high temperature, perhaps tonsillitis, bronchitis, or some similar affection. We admit very few cases of acute delirium here, but I notice a much higher temperature in such cases than in acute mania. The temperature rises as high as 103.5 F."

The most elaborate and interesting series of charts which I have received were sent by Dr. H. A. Tomlinson, superintendent of the St. Peter State Hospital of Minnesota. In this series are included—eighteen cases of acute mania in which the temperature ranged from 98° to 100.5°, except when there were distinct complications such as tonsillitis, erysipelas, etc.; only rarely in these cases was the temperature up to 100°: two cases in which without any apparently sufficient cause the temperature went up to 102°: one case in which two hours after the entrance into the hospital the temperature reached 102.2°, the rise probably having been due to excitement of coming to the hospital, especially as there were retention of urine and constipation (the day after, the temperature fell to 100.5°): one case in which without any other complication than constipation the temperature reached 102.8°, the case ending in complete recovery: one case in which the temperature reached 101°, with the result of final recovery: one case in which the temperature was almost all the time below 100.5°, but without obvious cause once went to 102.2°. (At the last report this case was not improving.)

Contrasting with these cases of acute mania the charts sent by Dr. Tomlinson, marked acute delirious mania, we find that in one case the temperature ranged from 97.2° to 103.4°; in one case the temperature reached 106.7°, death occurring a few hours after entrance; in one case, in which death occurred on the twelfth day, the temperature was almost always below 101°, once rising to 104°, and one day reaching 106.5°; in one fatal case the temperature ranged from 98.5° to 104.5°.

So far these records of acute delirious mania contrast strongly with those of acute mania, but in other cases sent by Dr. Tomlinson the temperature sheets correspond very closely with some of those of acute

mania. Thus, in one case, finally ending in dementia, the range of temperature during the first nine days was from 99° to 101.5°; there was then erysipelatoid inflammation of the arm, with a rise of temperature to 105°; after this complication had subsided for many days the temperature ranged from 99° to 101.5°; this being followed by a very irregular temperature, which some days rose to 103°. In another case which ended in recovery the temperature for forty-one days ranged from 99° to 101.8°, going higher for a short time on two occasions, once directly after the patient was brought into the hospital, when the temperature reached 102.6°, and once when there was an attack of erysipelas, when the temperature mounted to 103.4°. In still another case, which also resulted in recovery, for five or six weeks the temperature was between 99° and 100.5°, shooting up, however, on two or three occasions without obvious cause to 101.8° and 102.4°.

These observations seem to me to be very strong evidence that so far as concerns the temperature no sharp fixed line can be drawn between acute delirium and acute mania. It may be argued that the diagnoses made by Dr. Tomlinson were not in these cases accurate. If this were true, it would go far to prove that clinically no positive, sharply defined diagnosis or diagnostic rules separating the two disorders are possible. The very numerous temperature sheets furnished by Dr. Tomlinson show at once how wide his field of experience and how carefully he gleans it. The result of our inquiry so far seems to be that no diagnostic difference can be made out which shall enable it to be said with positiveness in all cases: this is an acute mania; this is an acute delirium.

In attempting to discuss the pathology and nature of acute delirium it seems to me essential to get, in the first place, some solid ground under our feet and then to examine the general territory. I therefore now report the following case, which was studied with sufficient care and results to give a foundation in fact.

Mr. F. was a man of rather feeble and distinctly neurotic organization, with a tendency to worry excessively, both as to business and domestic affairs. August, 1893, he consulted Dr. Thomas Potter, who found evidence of nervous exhaustion without any very distinctive symptoms, and advised complete rest, which advice was not taken. Through the early part of September there was sleeplessness, with depression of spirits, and about the 18th or 20th Mr. F. began to be very suspicious and to express the belief that he was being watched by detectives. September 22d he was very nervous, and finally left the bank where he was employed, during banking-hours, and walked to his father's house, about six miles in the country, because, as he afterward said, he was afraid to go in the cars as he believed detectives were in the cars looking for him. On reaching home Mr. F. was in a very exhausted condition; during the following night he was sleepless, and declared to his wife that he saw lights in the windows and the faces of detectives looking into the room; finally, he got up and went down

stairs, starting to go out of the house, but shrunk back because he said he saw detectives at the windows and doors, also the flashing of bull's-eye lanterns on the lawn. He was seen by me in consultation September 25th; his conversation was entirely rational; he recognized completely that the things he had seen at night had no real existence, but affirmed that he had seen them with great distinctness, and that at the time he saw them he believed in their real existence. According to the father, there had been distinct signs of mental confusion in his talk, but he had not failed to recognize where he was or the personality of his friends, except once or twice at night. The temperature was 99.5°; pulse weak; tongue relaxed and coated. During September 26th, Mr. F., while alone in his room, began to pray out loud in a broken, interrupted manner, growing louder and louder in his tone until he was screaming at the top of his voice, when his father came into the room. Mr. F. then went into a spell of furious kicking and screaming, lasting ten minutes, and followed, according to the statements of those about him, by a prolonged muscular rigidity.

According to the reports of the nurse, during the day of September 27th he talked rationally, ate heartily, until eight in the evening, when he had a hysteroidal attack similar to the one the day before. September 28th, during the night, his sleep had been interrupted by delusions, which continued more or less into the day, though at times he seemed rational; toward evening he again became hysteroidal. September 29th, temperature taken at midnight, 99.4°. September 30th condition not much changed, except that he either would not or could not urinate, but had to be catheterized. The evening of the 30th he became wildly maniacal and excessively violent, so that it required the combined strength of three persons to restrain him; in an hour this passed into a condition of quiet moodiness. October 1st he was quiet and apathetic; when aroused his speech was incoherent and his mental functions much confused; this confusion was especially marked in the evening, and in the early night he had a sudden attack in which there was apparent unconsciousness, with widely opened eyes, dilated pupils, strabismus, clonic spasms of the right side, lasting about five minutes, followed by rigidity of the neck-muscles and clinching of the hands. A moment after this he sprang up on the bed, perfectly wild, dancing, shrieking, yelling, etc. After this time he never became rational except for about fifteen minutes on October 2d. On October 2d the axillary temperature was taken and found to be 100.2° after a paroxysm; later, 99.5°. Later, on October 2d, he talked incessantly, was irrational, refused food or to urinate. October 3d axillary temperature was taken once, 99.4°. He seemed to be getting weaker, and had one violent paroxysm in the evening; also, involuntary passages in the evening. October 4th I was requested to see him again; found him refusing food, violently maniacal, fighting, attempting to escape, struggling, requiring four men to keep him in bed. I had him removed the same day to the University Hospital. October 5th he was more quiet, but almost collapsed; much of the time there was a muttering delirium; once during the day he asked for a cup of coffee. October 6th he had irregular, alternating periods of semi-stuporous quiet, of muttering delirium, and of furious mania, with fightings and strugglings. He recognized me, however, in the evening, but could not talk rationally. The symptoms of weakness steadily increased. At 3 A.M., October 7th, evidences of collapse

became very marked; from this time forward he was much of the time stuporously quiet, but whenever aroused was violent; at 1 P.M. he died in coma.

From October 4th until his death the axillary temperature registered, with all kinds of irregularities, from 100.8° to 102.8°; most of the time it was between 101.5° and 102.2°.

At the autopsy there was found extraordinary congestion of all the sinuses and membranes of the brain, with distinct serous exudation in the pia mater, and even some few very small fibrinous flakes. The dura mater, the base of the brain, and the bloodvessels were all apparently normal. Fluid was taken by Dr. A. C. Abbott, in charge of the Bacteriological Department of the Laboratory of Hygiene of the University of Pennsylvania, from whom subsequently the following letter was received:

"OCTOBER 10, 1893.

"DEAR DR. WOOD: I have not succeeded in finding *anything* either by microscopic or culture methods in the material taken from the meninges of your case on Saturday. Very truly yours,

"A. C. ABBOTT."

The following exhaustive report upon the brain was made by Dr. John Guitéras, Professor of Pathology in the University of Pennsylvania:

"The brain came under my observation after laying in Müller's fluid twenty-four hours. No gross lesion was apparent, except that the membranes were congested but not opaque. Any slight opacity present may have been made invisible by the action of the fluid. Fresh sections into the brain-substance showed well-marked congestion. The gray matter of the cortex was darker than normal, and the white substance well dotted over with red points. After hardening in Müller's fluid sections for microscopic examination were cut from the frontal lobe, the transverse convolutions, and the occipital lobe.

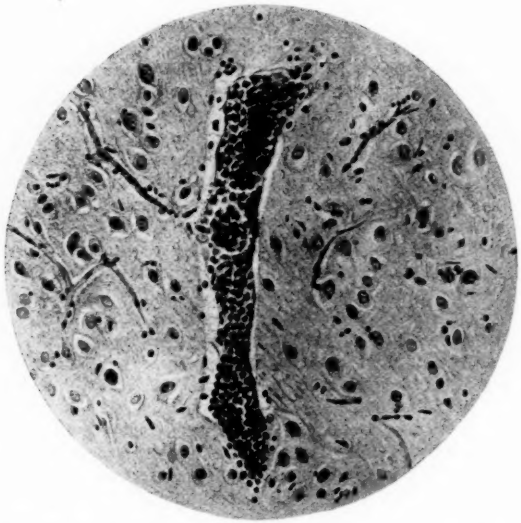
"The lesion found is an acute inflammation in the anterior portions of the brain. The inflammation is most marked in the anterior portions of the frontal convolutions. From thence it gradually diminishes until it is nearly absent in the occipital lobe. The inflammatory process affects the bloodvessels, the lymph-channels, the connective tissue. In these structures it is evidently a primary affection. The ganglionar system is not structurally affected in corresponding measure. In fact, the ganglion cells are generally normal, even in close proximity to vascular areas that are deeply involved. The features of this inflammatory process are peculiar, and may throw some light upon the question of the source of leucocytic infiltration in general.

"*Frontal lobe.* Vascular and lymphatic changes.

"Many of the pial vessels and of the cortex and white substance present a pronounced perivascular round-cell infiltration. This is perhaps best marked at the bottom of the sulci, but not always so. The inflammatory changes are not uniformly distributed. In the same microscopic field we find bloodvessels affected with others that are not. Some vessels may be traced, showing parietal cellular infiltration up to a certain point, and the remainder of the vessel is normal. Portions of the meninges over one convolution may show evidences of inflammation, whilst other portions are not affected or but slightly so. In the latter we notice that the perivascular spaces are wider than normal, and

present isolated lymphoid cells; in the former the accumulation of these cells may form bands along the vessel, of greater width than the vessel itself, as shown in the illustration. In close proximity to one another we find, then, all stages of this morbid process. This statement applies to the deeper portions of the cortex and to the white substance as well as to the meninges, though the cellular infiltration is never so great within the brain-substance as it is in some places in the pial covering.

"The bloodvessels affected are generally distended with blood. The capillaries and smaller vessels in the brain-substance are prominent features in the microscopic field; either because of their over-distention or because of the perivascular drift of round cells, or, as is frequently the case, for both reasons combined.



A bloodvessel in the deeper layers of the gray matter, engorged with blood, showing especially at the lower end clusters of round lymphoid cells.

"The round cells in the perivascular spaces are mononuclear lymphoid cells of the ordinary size; some very small ones are found, and some of the larger size.

"Though we have here an opportunity to study this process in all its stages, either by comparing the posterior with the anterior portions of the brain, or even within the areas of greatest development of the lesion, we do not see anywhere any preparation for an extravasation of leucocytes. There is nowhere to be seen a peripheral drift of leucocytes within the vessels, nor any accumulation whatever of such cells in the lumen of the vessel. It is impossible, then, to conceive that these vessels, containing nowhere (exceptions to be mentioned subsequently) any excess in the normal proportion of leucocytes, should be the source of the

enormous perivascular aggregation. It is very evident that the lymph-spaces in the walls of the bloodvessels are the primary seat of the cellular accumulation. When we do find, exceptionally, an excess of lymphocytes within a bloodvessel the appearances point clearly to an inwandering from the perivascular spaces. Besides the fact that in all stages the number of wandering cells is far greater outside than inside the vessel, we have direct evidence of the inward passage of such cells. The process takes place in the following manner: The lymphocyte exerts a peculiar influence upon the endothelial cells of the lymph-space and of the bloodvessel. The endothelial cell swells and its nucleus becomes paler. The swelling projects, extending around the lymphocyte in the same fashion as the uterine mucous membrane grows around the ovum. The lymphocyte is thus furnished with a coma of refracting protoplasm. This is carried forward into the vessel with the cell, bulging into the lumen. This coma is apparent around the lymphocyte even after it has penetrated the wall of the vessel, unless several cells are found together, in which case it almost disappears. In one instance a distinct track is left in the wall of the vessel, indicating the line of passage behind the corpuscle.

"It is interesting to note that the lymphocytes exert the same peculiar influence upon endothelial cells in other localities. This is seen at times in the lymphocytes that are found in the pericellular spaces of the ganglion cells. The change in the endothelial cells, however, is by no means general. Lymphoid accumulations are found frequently in contact with endothelial cells that are in all respects normal.

"There is nowhere to be found in these tissues any tendency to liquefaction or suppuration, such as would be shown by degenerative changes, by polynuclear formations, by very close packing of leucocytes, with a tendency to softening in the centre of the accumulations. On the contrary, we find frequently in the lymph-spaces where lymphocytes are grouped together fine protoplasmic prolongations that form a fine reticulum within the said spaces.

"No bacteria or other abnormal contents are found in the bloodvessels.

"The lymphocytes are found in excess throughout the reticular groundwork of the brain-substance. They are frequently found in contact with the Deiters's cells; and one, two, or three are found in many of the pericellular spaces of the ganglion cells.

"The Deiters's cells appear to be more numerous than normal, and large sizes are more frequent than in health.

"*The ganglion cells.* These do not show any degenerative changes. The pericellular spaces are large and contain frequently one or more lymphocytes. The ganglion cell is occasionally displaced by the manipulation, and its outline is consequently somewhat distorted. This appears to be due to the serous infiltration in the pericellular spaces. The protoplasm shows no structural changes. The nucleus stains in the usual manner and the nucleolus is distinct. There are no vacuolations. The granular reticulum of the nucleus is not so pronounced as it should be. Otherwise these ganglion cells appear to be normal.

"There is no evidence of degeneration of the myeline. The picture given by Weigert's stain does not differ from that of the healthy brain. In this case it is evident that the drift of lymphocytes has taken place along the lymph channels toward certain bloodvessels.

In regard to the pathology of acute delirium there is not an absolute unanimity amongst recent writers, but the condition is believed by Krafft-Ebing to be one of acute hyperæmia, with irritation, followed by exudation and symptoms of paralysis due to pressure upon the nerve-elements. The post-mortem appearances are well summed by R. Percy Smith in Tuke's recent *Dictionary of Psychological Medicine*, as consisting of "intense cerebral hyperæmia in the excited period and œdema in the later stage. The cellular elements are said to be swollen, opaque, granular, and to stain badly, and the pericellular and perivascular spaces to contain red or white corpuscles; and there are sometimes white lines along the vessels from extravasated leucocytes, and occasionally small extravasations of blood in the brain-substance."

It will be noted that in the case which I have just reported the pathological changes found by Professor Guitéras are accordant with the description of Dr. R. Percy Smith. I have once before seen a similar case with similar results at autopsy, and it seems to me that it must be considered as firmly established that there is a group of cases presenting symptoms of violent mental delirium ending in death, in which the lesion is an acute peri-encephalitis. That the acute peri-encephalitis is the essential lesion is strongly confirmed by certain clinical facts. As has been distinctly pointed out by Dr. George H. Savage (*Medical Press and Register*, 1891, vol. ii.) and other alienists, some cases of acute delirium pass on into general paralysis. The lesion of general paralysis is chronic peri-encephalitis, and it is natural that the acute disorder should sometimes pass into the chronic.

Further, it is well known that locomotor ataxia not rarely ends in chronic peri-encephalitis, just as chronic peri-encephalitis not rarely has locomotor ataxia as a secondary complication. A case which I reported some years ago demonstrated that acute delirium may develop in locomotor ataxia. The clinical and pathological facts agree in showing that there is a clinical and pathological relation between acute delirium and general paresis. The conclusion just reached does not in any way involve the further conclusion that *all cases* which present the clinical features of acute delirium, and are labelled by alienists and clinicians as acute delirium, are of the nature of an acute peri-encephalitis. It does not in any way account for the production of the peri-encephalitis.

In a paper read before the Philadelphia Neurological Society in 1890 Dr. J. H. Lloyd strongly argued in favor of the view that acute delirium is really toxæmic. It is entirely conceivable that a toxic agent may produce, by a direct action upon the cells, cerebral symptoms closely resembling those of acute delirium and at the same time superinduce typhoid symptoms. Again, it is entirely conceivable that a toxic agent may produce an acute delirious condition by provoking a peri-

encephalitis. For clear thinking, it is essential that the two methods of action of a poison be distinguished.

As above stated, analogy indicates that a peri-encephalitis of acute delirium may be produced in various ways. It is generally acknowledged that there may be a meningitis which is due to sunstroke or other cause not bacterial nor even toxæmic; but it is equally certain that a meningitis may be produced by some constitutional poison like that of sepsis or of epidemic cerebro-spinal meningitis. Just so, I believe, an acute peri-encephalitis may be produced by a poison or may be the outcome of an intense functional excitement or other cause not toxic.

There have been recorded a few bacterial studies of cases of alleged acute delirium. Those with which I am familiar have been gathered together by Dr. Charles K. Mills in volume iii. of the *Transactions of the Congress of American Physicians and Surgeons.* The most important case is that of Rasori—a case, however, in which the symptoms were not in absolute accord with those typical of acute delirious mania; the difference being in that the attack came on with persistent headache, irregular pupils, opisthotonos, and without fever. At the autopsy Rasori found in the sub-dural liquid a small bacillus, which he believed to be peculiar, and which injected into rabbits caused death with symptoms of acute septicæmia.

In a second case, reported by Drs. Charles S. Potts and Berlet, the symptoms occurred in a man who had used whiskey and morphine in excess, and who also was suffering from an open carbuncle. At the autopsy, in the brain cortex there were found perivascular exudation and leucocytes in the lymph sheaths and perigangliar spaces, and cultures from the cerebro spinal fluid made by Dr. D. Braden Kyle demonstarted the presence of the Streptococcus lanceolatus and also of the Staphylococcus pyogenes aureus and albus. Evidently this was simply a case of septic peri-encephalitis.

In another case reported by Dr. James R. Hunt, in which there was abscess of the kidney, Bacillus pyocyaneus was obtained from pure cultures from liquid in the lateral ventricle. Evidently, this again was a case of septic poisoning.

The only evidence that is at present forthcoming to show that there is a peculiar bacterial disease resembling acute delirium is that given by Rasori. In the case which I have reported in the paper no bacillic organisms could be found in the sections of the brain, and the very careful culture-experiments of Prof. Abbott demonstrated that the cerebral liquid was free from them. There was evidently no septic poisoning in the case, and it seems to me to demonstrate, as far as one case possibly can, the existence of a non-septic and non-bacterial acute peri-encephalitis. Moreover, the clinical history of the case, in the comparatively slow development of the mental symptoms, in their

coming and going, in the character of the delusions, and in the early absence of fever or constitutional physical evidence of disease, seems to me almost sufficient to prove that the attack was not bactericidal. A delusion-of-persecution bacillus may not be unthinkable, but it certainly is at present unknown; and it seems to me that the conclusion is fully established that there is what may be termed idiopathic acute peri-encephalitis, constituting at least one form of the so-called acute delirium.

I do not see how any physician can read the reports of cases which have been published as instances of acute delirium without having had the suspicion strongly aroused that under this heading have been described by various practitioners cases representing several affections. Of these diverse diseases which have thus been recorded under the one name, three types have already been considered in this article, namely, septicæmia with septic delirium, septic peri-encephalitis, and idiopathic peri-encephalitis. A fourth type seems to me to be easily discerned, namely, acute brain-exhaustion, or, in other words, a violent form of confusional insanity. It is probable that this type includes a very large proportion of the reported cases of acute delirious mania, as alienists of wide experience believe that all acute delirious mania is of this character.

Thus, in his letter replying to my inquiries, Dr. John B. Chapin, of the Pennsylvania Hospital for the Insane, writes that he believes that "acute delirious mania is an acute neurasthenia or the culminating of the active nervous exhaustion ending with a psychical explosion." Dr. John Curwen, of the State Hospital for the Insane at Warren, Pa., in a letter, after detailing two cases which he believed should be considered as acute delirium, and in which great relief and final recovery were obtained by free use of stimulants and feeding, says: "My own conviction has been that these cases were the result of great nervous and physical depression giving rise to the mental excitement, and were to be treated to meet these conditions in the most active and positive manner, or death would close the scene in about ten days."

That the term acute delirium or acute delirious mania has been used to cover various conditions, and that among these conditions is a peculiar cerebral exhaustion, are further shown by the following quotation from a letter written by Dr. H. A. Tomlinson, in which he says:

> Basing my opinion on the observation of a large number of cases, I believe the term "acute delirium" a better one than "acute delirious mania," because the condition is not in any sense one of mania, although it may be added to mania, and where it is not fatal and recovery does not take place mania may follow. Acute delirium has, in my experience, occurred in the course of all forms of insanity in their early stages, but it has been independent of the insanity, a condition *sui generis*. The natural history of this condition of delirium is very similar to that of the same condition in so-called "typhoid pneumonia," or the delirium of typhoid fever where the poison has spent its effect principally on the nervous system. Where this

delirium exists as *apparently* a primary form of insanity it has in my experience almost invariably been preceded by prolonged mental over-strain, shock, or physical exhaustion; and the immediate antecedents of the attack have been constipation, insomnia, exhaustion from refusal to take food, with a variable period of confusion accompanied by either exaltation or depression. In those cases dying within the first ten days we almost invariably find some degree of organic change in the kidneys or heart, besides the intense congestion of the brain and its membranes. While I was an assistant in this hospital three years ago I had seven successive cases of acute delirium, all of whom died within a week after admission. At that time, however, we had no trained nurses and the temperature-records were not accurate enough to warrant me giving them to you. In discussing these cases too little attention is given to the history of the individual before the onset of the delirum. My experience would bear out your suggestion that *clinically there are regular gradations from mania into delirium, and from delirium into mania.*

November 6, 1889, I read before the College of Physicians of Philadelphia a paper on "Insanity Following Acute Disease," in which I supported the proposition that these insanities constitute one disease-group having a uniformity of symptoms as characteristic and as complete as is usually seen in mental diseases, and resting upon a common pathological basis of disordered brain nutrition and exhaustion without anatomical alteration sufficiently gross to be detected by our present methods of study. In accordance with this proposition it was proposed to call all these cases "Confusional Insanities," including in the group the "stupidity or primary curable dementia," of Krafft-Ebing, the "Wahnsinn" of the same author, and the "delusional stupor," "mania hallucinatoria," and "confusional insanity" of various writers. Without discussing in detail, it may be well to summarize the proofs of identity of these affections.

1. If the cases vary much in their details, the general scope of symptoms and the general course of the disorder are identical: there are always mental confusion, hallucinatory excitement, loss of mental power, with similar constitutional symptoms; the cases nearly always ending in complete recovery if there have not been pre-existing organic disease of the kidneys, arteries, or other vital organs, and when death occurs there is similar absence of lesions in the cerebral cortex.

2. A similar mental condition with similar symptoms is produced by starvation and exposure, as in shipwreck.

3. The belief that the insanity has a specific relation to the poison of the disease which it has followed involves the absurd proposition that there occur as sequela to various acute diseases a large number of distinct specific insanities which offer the same general symptoms, pursue one general clinical course, and, when death occurs, agree in their pathological results; it being, moreover, possible to have these alleged distinct insanities produced not only by diseases which are connected with the presence of bacteria or other poisons in the body, but also by the shock of surgical operation or injury at a time when there is no septic poison

in the system, and even by overwhelming emotional disturbance during a healthy state of the organism.

In the short time which has elapsed since the publication of my paper upon confusional insanity a number of articles have been published. The following notes upon these papers seem to me of interest as showing in a short space the general drift of medical opinion and the difficulties which lie in the way of a clear perception as to the truth of the subject.

Dr. Henry M. Hurd (*American Journal of Insanity*, vol. xlix.) appears to believe that there are three distinct insanities following acute disease, respectively due to shock, to specific poisons, and to anæmia or nervous exhaustion. He illustrates the first of these causations by a case which followed removal of the ovaries; the second by a case which occurred after pneumonia; and the third by a case following typhoid fever. As I read these reports they do not seem to confirm the etiological theories of Dr. Hurd.

In an article on acute delusional mania, Dr. G. W. McIntyre (*Northwestern Lancet*, vol. xiii.) differentiates acute delirium by the suddenness of onset, the violence of the excitement, the rapidity of the exhaustion, and the presence of a febrile reaction in which the temperature follows no definite course. He says it is commonly produced by shock or exhausting physical disease when the temperature gets as high as 103°.

Dr. Alex. Robertson (*Journal of Mental Science*, vol. xxxvii.) finds the essential symptoms of acute dementia to be rapid failure of mental power with hallucinations; rapidly developed stupor; emaciation; feebleness of circulation; coldness and blueness of the extremities; brown tongue, etc. It is worthy of note that Dr. Robertson has obtained good results by the application of a double skull-cap containing hot water (110°).

Mr. C. Norman (*Trans. Royal Acad. of Medicine*, Ireland, 1890, vol. viii.) states that Korsakoff, of Moscow, thinks that alcoholic neuritis often exists in connection with confusional insanity from alcohol. Norman believes that the insanity which is described by a patient's friends as having "come on out of sleep" is always confusional insanity. He quotes Salgo as saying that any case in which hallucinations occur must be rejected from simple mania, and distinguishes confusional insanity from acute mania by absence of exaltation and increased rapidity of thought—from acute dementia by absence of complete stupor.

In an elaborate paper, Dr. John Ferguson, of Toronto (*Alienist and Neurologist*, vol. xiii., 1892), gives analyses of large numbers of papers upon insanity after surgical operations and fevers, and records a number of cases. There does not seem to be much in these papers necessary to comment upon, except that the idea is brought forward by several of the writers that the insanity is really septic. Dr. Theodore B. Hausen says "it is scarcely an exaggeration to say that if in the first week of the puerperium a psychosis in the form of an acute confusional insanity with

hallucinations appears, without the occurrence of another non-puerperal acute infectious disease, and without preceding eclampsia, one can be sure puerperal infection is present, even when fever and other physical symptoms are not discoverable by a thorough examination." Clouston also seems to be in favor of the septic theory; and Olshausen divided puerperal psychoses into: 1, those after septic conditions, the infection-psychoses; 2, those after post-partum hemorrhage and during lactation, the exhaustion-psychoses; 3, those following eclampsia, when there is uræmia or some other impurity of blood, the intoxication-psychoses.

The differences of opinion in regard to the question of confusional insanity which are shown in the abstracts just given, and which certainly exist among alienists, seem to me to be largely due to the failure on the part of many to apprehend certain thoughts and generalizations. It is plain that in septicæmia there may be a delirium which may simulate a form or stage of confusional insanity, although it is not dependent upon any disease of the brain cortex, but is due to the direct action of the poison or poisons in the blood upon the cerebral nerve-cells. What is true of septicæmia is undoubtedly true also of uræmia. As septicæmia and uræmia frequently occur during the puerperium, puerperal septic and uræmic deliriums are far from rare phenomena.

Further, the cases which I have abstracted from the article of Dr. Mills prove definitely that there is such a thing as septic peri-encephalitis; so that it cannot be gainsaid that after childbirth either septic delirium or septic peri-encephalitis may occur, and may very well be spoken of as puerperal mania.

Cases of the characters just spoken of constitute evidently the infection-psychoses and the intoxication-psychoses of Olshausen. Their existence in no way disproves the occurrence during the puerperal state of true confusional insanity. I suppose the exhaustion-psychoses of Olshausen include cases of the latter character; but it would be a mistake to suppose that the puerperal confusional insanity must necessarily be preceded by any apparent great exhaustion or by any violent extraordinary cause of exhaustion like a post-partum hemorrhage. The emotional and physical disturbances of childbirth may well produce a confusional insanity, since they are in many cases incomparably more severe than minor operations, such as excision of the breast or the sewing-up of a ruptured perineum, which are occasionally followed by mental disease.

Moreover, although the condition of the brain-cells which underlies confusional insanity may be produced by other than toxic causes, it seems to me indisputable that it may also be produced by toxic disturbances of nutrition. This is well seen in delirium tremens, a disorder which is getting more and more to be recognized as a variety of confusional insanity. The mental disturbance of delirium tremens

cannot be due to a direct action of alcohol in the blood or in the nerve-tissues at the time of the breaking-out of the disease. The "horrors" of the drunkard is the beginning of delirium tremens, and if the cerebral manifestations of the horrors or of the delirium tremens were due to a direct action of the alcohol, then they should be intensified, not relieved, by further doses of the poison. Evidently the symptoms are the result of nutritive changes in the ganglionic protoplasm which have been produced by the poison. In other words, the symptoms are only indirectly caused by alcohol. As acts alcohol, so in all probability may septic poisons act; as there is an alcoholic delirium and also a confusional mania which we call alcoholic, because it is indirectly caused by alcohol, so also are there in all probability a septic delirium and a confusional mania which is the secondary result of septic disturbance of nutrition.

The variations that occur in the symptoms of confusional insanity are certainly great, but does not almost every disease vary symptomatically? I suppose every practitioner of medicine has followed cases to the end in which after recovery he has been at a loss to say whether he had or had not to deal with such a comparatively simple affection as typhoid fever. Considering the complexity of the brain, the symptomatic variations in confusional insanity are not greater than are those of typhoid fever. More than this, owing to the fact that exaltation and depression of the cerebral cortex produce such similar symptoms, some cases of confusional insanity can scarcely be distinguished clinically (without the history of their whole course) from acute peri-encephalitis, and yet post-mortem examination shows that the lesion of the brain is essentially diverse. As proof of this fact I append the history, with autopsy, of a case in which the insanity followed the operation for laceration of the perineum, a case in which there were no symptoms of septicæmia at any time, a case in which the perineum had completely healed before the mental aberration became pronounced, and many days before the first febrile symptom was manifest, a case in which the autopsy proved the absence of any inflammatory lesions of the cortex. The contrast of this case with the one previously reported in this paper is sufficient to show how two cases of brain-disease closely resembling one another may have entirely diverse lesions.

Mrs. M. K., aged fifty-five years, widow, with one child living. Operated upon by Dr. Goodell for laceration of the perineum, December 2, 1892. Operation required about one hour, but the temperature during the following days did not rise above 99.6°. Ten days after the operation she voided urine naturally for the first time. The bowels were costive, but there were no other complications, and the mental condition of the patient appeared to be normal, though she seemed a little quiet. Twelve days after the operation it was noticed that the mental condition was changing, the patient becoming morose and

refusing to take food. During the next five days her mental condition became more decidedly affected; she became suspicious, believing that the nurse and the patients in the ward were plotting to injure her son and to interfere with her own money affairs; even her son could not pacify her, and at times she appeared not to recognize him. She progressively became more and more disturbed, getting out of bed, insisting on walking around the ward, not recognizing where she was, moaning and wringing her hands, and at times talking rapidly and incoherently. Her temperature during the last five days before she came under my care, on the twenty-first day after the operation, varied from 98° in the morning to 99° in the evening; the pulse from 72 to 92; the respiration was steady at about 20; the sleep was much broken, and the patient very frequently got out of bed at night; the bowels were constipated, but were kept open with Rochelle salt.

The abstract of notes taken after her transference to my ward is as follows:

Twenty-second day after the operation. Very noisy last night, had to be removed from the ward. Her speech is now incoherent most of the time; she talks incessantly, and when she can be understood it is concerning some one persecuting her son. When aroused actively she answers questions rationally, but immediately rambles off into a confused babble. She is very restless, tries to get out of bed whenever she is left alone, but has brief snatches of sleep at intervals. Urine scanty, darker in color than normal; specific gravity 1025; slight trace of albumin, no casts.

Twenty-third day. Under anodyne patient slept quietly most of the night. The treatment is nourishment by liquid food as much as she can be made to take, but she fights and refuses to take food. Hyoscine, gr. $\frac{1}{100}$; morphine, gr. $\frac{1}{6}$; chloral, gr. 10, every eight hours.

Twenty-fourth day. Patient about in the same condition; under the narcotics, which have been varied *pro re nata*, she has been quiet. She has taken large quantities of milk and eggs, with stimulants.

Twenty-fifth day. Patient in the early part of the day very restless, but slept quietly for several hours after a hypodermic injection of hyoscine and morphine; then passed into a semi-comatose condition. During the day she was quiet except when aroused by feeding or other procedure, when she showed some signs of irritation; no paralysis, no disorder of the pupils, no stiffness of the muscles of the neck, and no evidence of local brain irritation; as the day progressed the stupor deepened. Food was taken better, and apparently digested very well. The mucous membrane of the mouth became very dry, the tongue fissured and was heavily coated, the breath very offensive. Urine passed involuntarily. When left to herself was absolutely quiet, but when disturbed would groan and toss about.

Twenty-sixth day. Mental condition about the same. Urine examined: specific gravity 1025, contained pus, albumin, and casts, hyaline and pale granular. Digitalis and stimulants were used freely. Toward night a decided change took place; the respiration became frequent, the face very pale, and the coma more marked.

Twenty-seventh day. Condition about the same. Repeated careful examination of lungs failed to detect any physical signs of pneumonia.

Heart-sounds normal. At times she is quite restless, tossing her head from side to side, flexing her legs, etc.

From this time until death, which occurred on the twenty-ninth day, the pulse gradually failed, and toward the last the whole surface of the body became cyanosed. Digitalis and strychnine were given hypodermically, liquid food and whiskey freely.

With much difficulty an autopsy was procured. On opening the skull there appeared to be some excess of blood in the sinuses, but there were no gross lesions either in the brain membranes or the brain-substance. The brain was put in Müller's fluid, and after hardening was given to Professor John Guitéras, who reported upon it as follows: "Sections for histological examination were cut from the frontal lobes, the transverse convolutions, and the occipital lobe. The result of this examination is negative. A number of sections from the several areas is submitted. They represent fairly well healthy brain-tissue. The methods of staining employed were ammonia-carmine, gentian-violet, hæmatoxylin, and eosin, and Weigert's myeline stain."

Unfortunately, in the case just reported no bacterial cultures were made, and, in so far, the record is imperfect. The history of the case, however, makes it highly improbable that there was any bacterial poisoning. The symptoms during life were those of confusional insanity, and in that coincided entirely with the etiological history. It is also unfortunate that a portion of the brain was not hardened in osmic acid, as possibly some lesions of the ganglionic cells might in this way have been made out. Nevertheless, the record is sufficiently complete to show that there was no peri-encephalitis and that there was no lesion in that brain discoverable by the ordinary methods of investigation. The case was evidently one of confusional insanity.

In attempting to sum up the result of our expiscation of acute delirium it becomes apparent that the deductions to be drawn are of two kinds: first, those which appear to be certainties; second, those which appear to be probabilities. The conclusions which seem to be established are: That cases of the so-called "acute delirium" are of various characters, representing several different diseases; that leaving out of sight septic, uræmic, and other intoxications, cases of which have undoubtedly in the past been recorded as acute delirium, there are two distinct diseases described as acute delirium—first, *acute peri-encephalitis;* second, an acute affection, probably primarily centred in the ganglionic cells, but without lesions that can be demonstrated by our present process.

Of the *acute peri-encephalitis* there are, again, several forms: one especially due to the presence of septic organisms in the brain itself, and therefore to be designated as acute *septic peri-encephalitis;* one in which there are no organisms, in which the cause appears to be emotional strain or other functional excitement, and which may therefore be known as acute *idiopathic peri-encephalitis.* If the observations of Rasori be correct, there must be a third peri-encephalitis, due to the presence of a

special organism, and therefore worthy of rank among the peculiar fevers or germ diseases.

The conclusions which are to mind probably, but not firmly, established are, that all manias of an acute type which are not intoxication-neuroses and are not due to the presence of organisms in the blood, are divisible into two affections: first, mania proper; second, confusional insanity, and that each of these diseases becomes, when in its most severe form, an acute delirium. Thus, there would be, first, *acute mania* —that is, mild acute peri-encephalitis, known when in its severest form as *acute delirium*—that is, violent, usually fatal, peri-encephalitis; second, *confusional insanity*, without demonstrable lesion, but probably the result of changes in the ganglionic cells themselves, constituting in its severest form an *acute delirium*, also without demonstrable lesion, but, in fact, due to an exaggeration of the unknown ganglionic or other alteration present in the confusional insanity.

UNIVERSITY OF PENNSYLVANIA, February, 1895.

A CASE OF MIDDLE MENINGEAL HEMORRHAGE. OPERATION; RECOVERY.

AN ANALYSIS OF FORTY-EIGHT CASES OF UNCOMPLICATED INTRA-CRANIAL HEMORRHAGE.

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I.

THE patient, a man, was brought to the Massachusetts General Hospital December 27, 1892. Examination found œdema of the right temporal region. Unconsciousness present. An interval of consciousness was absent. Slight bulging of the right eye. (See Case XXIII. of the Extra-dural Group.)

Operation in the right temporal region. A skin-flap was made over the fracture and œdematous area. A fracture was detected running from about the middle of the temporal ridge an inch back of the coronal suture outward and forward across the squamous part of the temporal bone to a half-inch behind the pterion.

The bone anteriorly to the fracture was depressed. The trephine was applied over the depressed portion behind the coronal suture. Upon exposing the dura no pulsation was seen. The dura was dark in color. A slight amount of extra-dural blood escaped. On following the fracture down to the base of the skull the dura was found lacerated, the anterior branch of the middle meningeal artery torn, and blood-clot and

lacerated brain-tissue present. The anterior branch of the middle meningeal artery was tied and the hemorrhage ceased. The blood-clots were removed, the exposed area cleansed with boiled water, and gauze drainage introduced. All the gauze was removed in four days. No unusual symptoms attended convalescence. Recovery was complete in three months. (See Fig. 1.)

FIG. 1.

This case is of interest because no fracture was detected before the operation, and it was supposed that the bulging of the eye indicated an increase of intra-cranial pressure, which proved true.

The method of operating was comparatively simple, in that the fracture was followed down until the bleeding vessel was found. This necessitated the free removal of bone below the trephine-opening.

There was no interval of consciousness in this case, and the conditions found easily explained its absence. The man was suffering from concussion and laceration of the brain as well as from intra-cranial pressure, and the interval of consciousness was obscured by the presence of the concussion. The recognition of an interval of consciousness, as will be seen by the analysis of the following cases, is of very great importance. If, however, the interval of consciousness is not present, as in the case reported, intra-cranial pressure from hemorrhage cannot be said to be absent, for concussion attendant upon the injury may mask the interval of consciousness which might have been present had the injury been less severe.

In this paper an attempt has been made to present for consideration in a concisely accurate form the subject of intra-cranial hemorrhage. The material upon which this study is based has been collected from French, German, and English medical literature covering the period from 1886 (the date of Jacobson's[1] exhaustive study) to September, 1894. Several cases of intra-cranial hemorrhage are here recorded for the first time, occurring at the Massachusetts General Hospital, including the case recorded above, which came under the immediate care of one of the writers, and was the direct stimulus to the present investigation.

It may with truth be said that intra-cranial hemorrhage due to traumatism and all "injuries of the head affecting the brain are difficult of distinction, doubtful in their character, treacherous in their course, and, for the most part," were formerly "fatal in their results."

To-day, because of a keener interpretation of the nervous symptoms and a knowledge of their localizing significance, because of aseptic methods of operating, and because of the improvement in operative technique, the mortality following intra-cranial hemorrhage is noticeably diminishing.

The importance of this subject is very great. This is shown by the frequency with which cases of this class are overlooked, by the slightness of the violence which produces so fatal an accident, by the suddenness with which death often occurs, and by the very great difficulty of differentiating between symptoms of the lesion under discussion and laceration, and concussion of the brain.

In approaching a case of head-injury the history of the case, including the story of the original violence, is of value. Many head-injuries, which at the time seem simple and unimportant, may prove serious, and may produce ultimately fatal results, as in Cases IX. and III. of Group A, and Cases V. and XXI. of Group B, in which cases the hemorrhage was caused by a blow from a whip-handle, a fall down a few steps, a penknife-wound, and a blow from a billiard cue respectively.

Other cases occur in which the injury is pretty considerable and definitely localized, as in Group A, Cases V. and XV., in which the violence was from the kick of a horse and a blow from a crowbar.

More often the violence is due to falls, and usually to pretty serious falls, as in Group A, Cases XVIII., XX., XXI., and Group B, Case XIX., which were falls down a flight of stairs, or from the top of a freight car, or from one story of a house.

In this series of operated cases there were 27 of extra-dural hemorrhage.

[1] Guy's Hospital Reports, vol. xliii., 1886.

The nature of the injury in these cases:

	Cases.
Falls	18
Blows	4
A pistol-wound	1
Unknown	1
Kicked by a horse	2
Blow with a cricket-ball	1

There were found to be 21 cases of sub-dural hemorrhage.

The nature of the injury in these cases:

	Cases.
Stab-wounds of head	2
Falls, striking on head	11
Blows on head	7
Unknown	1

The sources of intra-cranial hemorrhage which are of practical importance to the surgeon are:

1. From the middle meningeal artery and its branches;
2. From the veins of the pia mater;
3. From the sinuses of the brain;
4. From lacerated brain-tissue.

These four sources of traumatic intra-cranial hemorrhage cannot be easily differentiated clinically. The difficulty of distinguishing hemorrhage from any one of the sources from the associated laceration and concussion of the brain is at times extremely great.

The only very important *general* symptom of intra-cranial hemorrhage, excepting shock, is the interval of consciousness. An interval of consciousness often exists between the time of the injury or stunning of the patient by the accident and the onset of the symptom of compression due to the intra-cranial hemorrhage.

This interval of consciousness is not always present, and it may not be well marked.

In 21 cases of sub-dural hemorrhage here reported the interval of consciousness was present in 9 cases. Its presence was unrecorded in 1 case, and it was absent in 11 cases.

In 27 cases of extra-dural hemorrhage the interval of consciousness was present in 14 cases and absent in 13 cases.

In the sub-dural group the interval of consciousness existed in the following cases: II., IV., VIII., IX., XIII., XVI., XVII., XVIII., and XIX., and a detailed account is here given of these symptoms in each instance.

In Case II., two months after the fall, paraplegia and left hemiplegia appeared, and ten days later coma came on rapidly. In this instance the interval of lucidity was an extreme one and was an important factor in the diagnosis of the lesion. It would be interesting to know the path-

ological condition present which was the occasion of the hemorrhage at so late a date.

In Case IV. there was present a stupid, drowsy condition, which later (after seven days) became unconsciousness. This is not unusual. In place of an interval of clear mental activity drowsiness may precede the coma of great intra-cranial pressure. This should awaken alarm.

Case VIII. illustrates the typical interval of consciousness following a head-injury. A blow on the head; unconsciousness temporary; walks a good distance; becomes stupid or drowsy, and forty-eight hours after the receipt of the injury is semi-conscious; seventy-two hours after the injury is comatose.

Case IX. After a fall, which was forgotten; on the following day was drowsy; drowsiness continued; on the sixth day coma.

Case XIII. was stunned, but recovered consciousness almost immediately. On the third day after the accident delirium appeared followed by unconsciousness on the fourth day.

Case XVI. The interval of consciousness in this case was not well marked, although present; that is, after an injury unconsciousness existed, and ten hours after the accident the patient became partially conscious. It seems for all practical purposes that the presence of consciousness is relative, but it would be better, perhaps, for purposes of classification to make the distinction a perfectly clear and definite one; either the person is wholly conscious or is unconscious.

Case XVII. At first stupid and dull. This dulness increased up to thirty-six hours after the accident, then for two days there was improvement. He was discharged home, against the advice of his physician, and was returned in four hours comatose.

Case XVIII. An interval of three weeks of consciousness, followed by sudden unconsciousness and associated with general convulsions.

Case XIX. fell; walked home; later there was unconsciousness in varying degree through several days, which, because of its increase, indicated operation.

In the extra-dural group an interval of consciousness existed in the cases numbered III., IV., V., VIII., IX., X., XIV., XV., XVI., XVII., XIX., XX., XXI., and XXVII. In Cases XIX. and XX. the interval of consciousness was only partial. Reference may be had to the individual cases for an account of the symptoms in the extra-dural group.

Taking the two groups together, in the 48 cases of extra and sub-dural hemorrhage, 23 presented an interval of consciousness—that is, nearly one-half of all the cases.

Obviously there must be a large group in which the interval is never present, the concussion caused by the accident passing over into the coma of compression.

The duration of the interval of consciousness may vary from a few minutes to several weeks, as illustrated in the following two cases.

Case XVII., extra-dural group. The patient fell into the water, striking his head on a pile. He walked toward home, and in twenty minutes fell unconscious in a convulsion; spasms began on the left side of the body and appeared later on the right side. There was a scalp wound one and a half inches above the left mastoid process. The bone was not fractured. The breathing was labored. Pulse was about 60. Bleeding from the right nostril. Trephined one and a half inches behind the external angular process of the frontal bone on the right side. Four to six ounces of blood were removed and recovery was complete.

Case II., sub-dural group. A fall; two months later paraplegia followed by left hemiplegia. Ten days later tongue turned to the right. Thickness of speech, involuntary micturition, coma. Trephined over the right fissure of Rolando. Sub-dural clot found. Gradual and complete recovery.

The duration of the interval of consciousness depends on the number and the size of the vessels bleeding and the rapidity of the hemorrhage. If the patient is kept quiet and no stimulants are given, the hemorrhage will be less severe than if the patient is allowed to move about and freely stimulated.

Case XVII. of the sub-dural group illustrates very well the harm which may come from the patient's moving about. A man knocked down in the street was conscious but dull. At the end of thirty-six hours the stupor increased, but during the next two days the improvement in his mental condition was so great, and there being no localizing symptoms, operation was thought inadvisable. The man left the hospital against advice, and was returned four hours later restless and delirious. There were convulsions of the left arm, head was thrown back, right arm was rigid, respiration was labored and thirty to the minute; right pupil dilated, left contracted. Complete unconsciousness soon appeared, followed by coma and death four days later. [The autopsy is reported with the details of the case.] There can be very little doubt that the moving of this man to his home was the immediate cause of starting up fresh hemorrhage.

It may be impossible to detect the interval of unconsciousness because of the drunkenness of the patient.

Jacobson concludes that the interval of lucidity is absent in one-third of the cases.

Hutchinson says: "It would seem that it is not usual for a large hemorrhage to occur at the time of the injury, for almost always there is an interval during which the patient is well before the symptoms of compression come on."

The interval of consciousness being the only general distinctive symp-

tom of intra-cranial hemorrhage, it is necessary next to inquire as to the local symptoms—and one must consider the condition of the limbs as to hemiplegia, paraplegia, and rigidity; the condition of the pupils, whether they react to light and accommodation, and whether they remain of equal size; the character of the pulse; the character of the respiration; the condition of the scalp; whether, if unconsciousness is present it increases and passes into coma.

In the 48 cases on which this paper is based nervous symptoms were absent in only 5 cases. The variety of cerebral symptoms noted was aphasia, paralysis of one arm, paresis of one leg, paraplegia, spasms of the face and leg, turning of the eyes, spasm of the sterno-mastoid muscle, anæsthesia of different parts, rigidity, general convulsions, ankle-clonus, hemiplegia.

These cerebral symptoms may be well marked; they may be but little noticed—that is, temporary; certain of them may be more marked in one limb than in the other, or entirely absent.

The condition of the pupils. Nothing new was gained by a study of the pupils in these cases. Jacobson's observations were confirmed. If the pupils are natural as regards reacting to light, the compression of the brain is probably in a recoverable condition if trephining is immediately performed. If the pupils are insensitive and often at the same time dilated, the compression is probably extreme; and while trephining is demanded, it is improbable that the brain in these cases will recover itself after removal of the clot.

The Hutchinson-pupil is of the greatest importance. By this is meant a widely dilated pupil on the side of the injury—*i. e.*, opposite the paralyzed side, the other being natural or contracted. This sign, taken in connection with other evidence, is of supreme value. It indicates pressure on the third cranial nerve, and locates the pressure in the middle fossa.

The pulse. In cases of well-marked compression, uncomplicated, the pulse will be slower than normal and full. In cases of doubtful diagnosis it is important to record the pulse at frequent intervals—*i. e.*, hourly or half-hourly, that any variation may be quickly noted.

Unconsciousness: coma. Where the hemorrhage is rapid and the compression great the coma may be very deep at the outset. The degree and course of the unconsciousness are of importance, for a distinguishing sign of hemorrhage from one of the sinuses is the sudden coma, the pressure being severe. One of Dr. Elliot's cases here tabulated illustrates this point well. The beginning coma must not be taken for a natural sleep.

Respiration. In this group of cases in only nineteen out of forty-eight is there a record of stertorous breathing. The character of the breathing is more often omitted than recorded in the reports of cases. The Cheyne-Stokes breathing is noted in a few cases.

The condition of the scalp and cranial bones. Every patient suffering

from so severe a head-injury as to make it probable that trephining is likely to be done should have the head shaved of all hair. This will expose the scalp to good view. Ecchymosis and a feeling of contusion in the scalp—puffy swelling—will suggest an underlying fracture. A progressive intra-cranial hemorrhage may force its way through a fracture in the skull and give rise to a hæmatoma under the scalp.

In a given case of head-injury, having had no regard for the signs above mentioned, there may exist doubt as to the presence or absence of laceration of brain-tissue. The following facts may aid in a definite diagnosis: In middle meningeal hemorrhage the scalp-wound or bruise is usually over the course of the artery. The violence leading to middle meningeal or other hemorrhage may be very slight, as in certain cases already mentioned. An injury causing brain laceration will almost always be severe. If the concussion is severe, the interval of consciousness is apt to be absent. Where the brain is lacerated there are usually present twitchings and spasms of certain groups of muscles.

The treatment is, of course, in these cases trephining and the removal of sufficient bone to enable one to evacuate the blood-clot and arrest the hemorrhage.

If there is fracture present, the trephining should be in the line or fracture. If there be no fracture, the trephining should be, it seems to the writers, according to the rule of Voyt and Beck, at a point one and one-half inches behind the external angular process of the frontal bone, and one and one-half inches above the zygoma, which will locate the anterior branch of the middle meningeal.

Krönlein suggests the advisability of trephining twice, the first point being that suggested by Beck, the second point in Beck's horizontal line below the parietal eminence.

The size of the trephine-opening is determined by the demands of the case. A large trephine will not cut evenly through the bone on account of the curvature of the skull. It is best to use a small inch or inch and a half trephine and enlarge the opening with the rongeur forceps. Whether the dura shall be opened or not is to be decided in each instance on the general principle that it adds little to the risk of the patient, and, if unopened, much valuable information may be lost. The fact should be kept in mind that only in tumors of the brain are there liable to be herniæ cerebri after trephining. Strict asepsis should be maintained, and all the time taken that is absolutely necessary to an aseptic and thorough operation. In children a rapid operation is demanded.

The indications for immediate operation after the reception of an injury to the head are increasing paralysis, thickness of speech, persistent convulsions, an increase in any or all pressure-symptoms—*i. e.*, increasing unconsciousness—the Hutchinson-pupil being an important sign of increasing intra-cranial pressure.

Aphasia points to an involvement of Broca's convolution. Disorders of sight and hearing point to an extension toward the occipital and temporal lobes respectively. Paralysis of the third nerve points to an extension toward the base of the skull.

The mortality after operations on the head for intra-cranial hemorrhage other than cerebral hemorrhage proper has not been determined since 1886.

Jacobson in the cases studied previously to 1886 found that out of 70 cases of extra-dural hemorrhage 13 cases recovered from the operation, a percentage mortality of 81.43, and a percentage recovery of 18.57.

The group of cases here reported enables a fairly just estimate to be formed of the mortality to-day. Since 1886, then, of the 26 operated cases of extra-dural hemorrhage, 18 cases have recovered and 8 have died, a percentage mortality of but 30.77, and a percentage of recovery of 69.23. Of the sub-dural group of 19 operated cases, 15 cases recovered and 4 cases died, showing a mortality of 21.06 per cent. and recovery of 78.94 per cent.

In estimating the value of these figures it must be borne in mind that this is a group of selected cases; many cases have been operated probably which have died and have remained unreported. There must be a very few, on the other hand, which have been operated and recovered, and have been also unreported. These figures represent the mortality and recovery rates of only operated cases—that is, cases in which there have appeared sooner or later definite indications for interference. Of course, of the many unrecovered and unoperated cases there must have been many in which to-day there would have been discovered definite indications for operation. But after taking into consideration just what this group of cases is (here represented) the mortality is indeed low, and the percentage of recoveries is very encouraging for operative interference even in pretty doubtful and desperate cases of severe head-injury.

SURGICAL ANATOMY OF THE MIDDLE MENINGEAL ARTERY.

Steiner[1] has examined one hundred skulls with reference to this subject, with the following results:

In 57 cases the division of the artery into its anterior and posterior branches took place within the foramen spinosum, so that there was no main trunk of the artery within the skull.

In 35 of the remaining 43 cases the length of the main trunk within the skull was from 1 to 3½ cm. In the remaining 8 cases the length was 3½ to 5 cm.

In 38 cases the course of the anterior branch for a distance of 1 to 3

[1] Archiv für klin. Chir., 48 I. 101.

cm. along the anterior inferior angle of the parietal bone ran through a bony canal—*i. e.*, the vessel was completely surrounded by bone, so that it must necessarily be cut off by the crown of a trephine cutting through the bone at this point.

In 6 cases the anastomosis of the anterior branch with the ophthalmic artery, through the sphenoidal fissure, was so free as to constitute the true origin of the middle meningeal, so that either the posterior branch alone or even no vessel whatever entered the skull through the foramen spinosum, which in these cases may be entirely wanting.

A careful trial of the three recognized methods for locating the anterior branch for purposes of trephining was made with the following results:

Vogt's method locates the artery at the intersection of a vertical line drawn a thumb's breadth back of the frontal process of the malar bone and a horizontal line drawn two-fingers' breadth above the zygoma.

By this method the artery was exactly located in 43 per cent. of the cases. In the majority of the remaining 57 cases the point where these lines crossed lay behind the line of the artery. The point found by this method will vary with the thickness of the thumb and fingers of the operator, as is obvious.

Witherle's method locates the vessel at a point 1½ inches behind the frontal bone, and either one inch above the zygoma or 1½ inches above the articular process of the lower jaw.

By measuring from the upper border of the zygoma Steiner found that by this method he came upon the trunk of the artery in only 28 cases, and that in 65 of the remaining 72 cases the point so located lay from 1 to 2½ cm. behind the actual position of the artery.

Krönlein's method locates the anterior and posterior branches by first drawing a base-line through the lower margin of the orbit and the external auditory meatus. The anterior and posterior branches are both located on a line drawn parallel to this through the superior margin of the orbit, the first at a point 3 to 4 cm. behind the external angular process of the frontal bone, and the second at the intersection of a line drawn vertically from the posterior border of the mastoid process. By this method the anterior branch was located in 53 per cent. of the cases. In most of the remaining cases the artery lay in front of the point located. The anterior branch, or a large secondary branch, was located by this method in only 40 per cent. of the cases. Of these methods, that of Krönlein is certainly the best, since, admitting that an error of 1½ cm. is of no practical importance, the anterior branch will be located with sufficient accuracy in 70 per cent. of the cases.

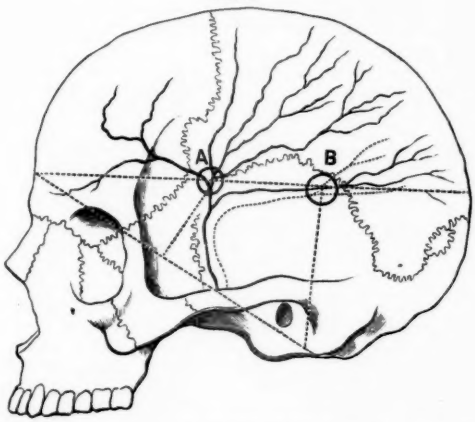
With reference to the anterior branch, all these methods have the disadvantage of locating the trephine-opening so low as to fall below the course of the artery in all the cases where it takes its origin from the

ophthalmic, and accordingly comes in close relation with the outer side of the skull at a somewhat higher point. A second disadvantage is that the artery is found by these methods at the point where it lies in a bony canal (as was found in 38 of the 100 cases), and is consequently liable to be cut off by the trephine.

A disadvantage of Krönlein's method for finding the posterior branch is that the trephine is applied in close relation to the transverse sinus. To obviate these disadvantages the writer suggests that the horizontal line be drawn at a somewhat higher level—namely, from the middle of the glabella.

For finding the anterior branch a line is drawn from the glabella to the tip of the mastoid process, and a perpendicular erected at its centre. The intersection of this perpendicular with the horizontal line will give the point for finding the anterior branch.

FIG. 2.



Method of locating the trephine-opening for finding the branches of the middle meningea artery. (After STEINER.) A. Point for anterior branch. B. Point for posterior branch.

The posterior branch is found at the intersection of the horizontal line and a perpendicular drawn just *in front* of the mastoid process. The author claims to have located the anterior branch in 90 per cent.

By this method it is obvious that the trephine-opening for the posterior branch is placed slightly above and in front of the point given by Krönlein's method, and therefore further from the lateral sinus.

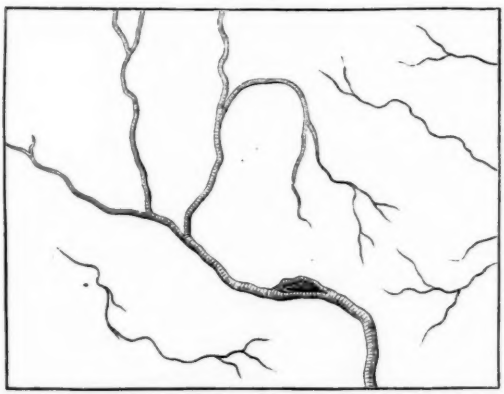
The writer goes on to state that exact methods of locating the artery are unnecessary in cases where it is possible to remove a flap of skin, muscle, and bone from the side of the skull, with knife and chisel, so as to expose the main trunk and both branches at the same time, either for tying the arteries or removing extravasated blood.

This method of osteoplastic resection is of course inapplicable (*a*) when the soft parts have been torn from the bone by the original injury, or extensively lifted from it by extravasated blood; (*b*) when compound fracture is present. In these classes of cases the exact methods of locating the artery will still be useful.

This method of osteoplastic resection, recommended first by Krause for gaining access to the Gasserian ganglion, was also suggested by him as applicable to injuries of the middle meningeal artery, which is exposed during the greater part of its course.

The flap is described by Krause as follows: "The base of the flap lies above the zygoma just in front of the tragus; the line then runs upward, curving backward at the same time; then describes a semicircle forward and returns to the zygoma, so that the base of the uterus-shaped flap is

FIG. 3.



Ruptured middle meningeal artery. The seat of rupture shows a hair lying in the artery. (Case, Warren Museum, Harvard Medical School, No. 7801.)

3¼ cm., height 6½ cm., and its greatest width at the upper part 5¼ cm." The writer recommends a similar-shaped but slightly larger flap, the width and height of which are each about 6 cm., and the width at the base from a thumb's breadth back of the margin of the orbit to the tragus. Near the anterior border of this opening the anterior branch of the artery will be found, and near the posterior border the posterior branch, and easy and free access will be gained to all hæmatomata in the temporal region. If the artery shall be found to have been ruptured still nearer the foramen spinosum, access to it can be gained by chiselling through to the zygoma and breaking off the lowest portion of the lateral wall of the skull. This method is said to have been successfully adopted by Professor Wolfler in two cases.

The following case and pathological specimen, illustrated in Fig. 3,

is extremely valuable as demonstrating a local lesion in the middle meningeal artery beneath a fissured fracture of the skull.

A young man was standing on the sidewalk, and was struck in the head by a brick. He fell, striking the sidewalk; recovered in a few minutes and was able to walk to the house of a friend, where symptoms of compression came on, and he died shortly afterward.

At the autopsy made by the medical examiner, Dr. F. A. Harris, of Boston, there was found a slight linear fracture of the skull crossing the course of the middle meningeal artery, which was ruptured. An extensive extravasation of blood had taken place between the dura and the skull. The convolutions were markedly flattened. The specimen is No. 7801 of the collection in the Warren Museum, Harvard Medical School. (See drawing, Fig. 3.)

Group of Extra-dural Hemorrhage Cases.

Case I. (Krönlein, *Deutsche Zeitschr. für Chirurgie*, 1886.)—A man, thirty years old, fell from a step while drunk, and was unconscious from the time of the injury. Aphasia. Left facial paresis and ocular spasm. Motor paralysis of the right leg and arm. Could be partially roused. Pupils contracted but react. Pulse 120, of medium strength. Respiration 28 to 30, superficial. Slight excoriation of skin in the left temporal region. On the eighth day stupor increased to coma, and right facial paralysis appeared.

Trephined on the left side over the anterior branch on supra-orbital level, 4 cm. behind external angular process of the frontal bone, and later, over the posterior branch of the middle meningeal. Hæmatoma, 240 grammes, filled the middle fossa and the posterior fossa as far as the occipital protuberance. A fissure was found running vertically downward in the temporal region, cutting off the main trunk of the middle meningeal artery. Answered questions in two days, and paralysis disappeared in three days. Recovery.

Case II. (Krönlein, *Deutsche Zeitschr. für Chirurgie*, 1886.)—A man, aged forty years, fell down some steps, and was unconscious from the time of injury. Moved when loudly spoken to. Three days later pulse 74; less movement of the left arm and leg on needle-puncture than of right arm and leg. Aphasia. Left abducens oculi paresis and nystagmus. Boggy area over right ear and slight tenderness on slight pressure over this same region. On the fifth day was still unconscious and the pulse was 120.

Trephined over the anterior division of the right middle meningeal. Dura punctured. No hæmatoma found. Death in twenty-four hours.

Autopsy. Fracture of the right temporal bone and parieto-occipital hæmatoma reaching forward within 3 cm. of the trephine-opening. A trephine over the posterior branch of the middle meningeal would have exposed this clot.

Case III. (Brunner, *Correspond. für Schweizer Aerzte*, June, 1888.)—A man, aged thirty years, fell from steps and was conscious for four or five hours; later, unconscious. Moved the left leg occasionally. Pulse 50 to 60, full and strong, but intermittent. Respiration stertorous, Cheyne-Stokes. Ecchymosis of the left eyelid. Both pupils were dilated and reacted but weakly. Right facial paresis. Œdema of the scalp in the left temporal region. Slight wound of scalp not reaching to bone. Right hemiparesis and hemianæsthesia.

Trephined over the anterior branch of the left middle meningeal. A fissured fracture running forward and including the groove for the middle meningeal artery was found, an extra-dural hæmatoma 3 cm. thick reaching forward to the frontal bone and toward the median line. Free bleeding from the posterior division and the anterior branch of the middle meningeal, which was checked by three catgut sutures. Hæmatoma, 200 grammes. Condition unchanged by operation. In twenty-four hours moved the right arm. Pulse 86

to 100. In forty-eight hours conscious, and moved right arm and leg. Died on the fifty-eighth day of pneumonia.

Autopsy showed a fracture of the base on the right side.

CASE IV. (Davies-Colley, *Lancet*, March 31, 1888.)—A man, aged fifty-three years, fell from a height of twelve feet and was stunned, but recovered consciousness, though remaining drowsy. There was paresis of the left arm. The pulse was slow and regular. A bruise was found in the right temporal region. On the eleventh day trephined over the right fissure of Rolando. A large extra-dural hæmatoma was removed. Recovery was complete.

CASE V. (Croft, *Lancet*, 1887.)—A woman, aged twenty-six years, was kicked in the head by a horse. There was an interval of consciousness which lasted several hours, when coma ensued. Twelve hours later there was still coma. Complete paralysis of the right arm and leg. A scalp-wound was found just below the left parietal eminence.

Trephined under this wound, and an extradural hæmatoma of two and a half ounces was removed, the hemorrhage being from the posterior branch of the middle meningeal. Moved the right arm and leg in six hours and spoke in two days. Recovered completely.

CASE VI. (Walker, *Medical and Surgical Reporter*, 1888.)—A man, aged thirty-five years, history not known. Stupor. Paresis of the left arm and leg and incontinence of urine. Pulse 40. Pupils reacted. On the second day the paresis had become complete and the unconsciousness had increased. On the fourth day the pulse was 38. There was feeble reaction of the pupils, unconsciousness still existed.

Trephined over the right fissure of Rolando. No fracture of the skull found. Extra-dural clot, four ounces. On the sixth day the patient had so far recovered consciousness as to be able to give the history of having fallen from a scaffold ten feet high. He worked three hours after the fall, walked part of the way home, and fell. Complete recovery in a month.

CASE VII. (Stokes, *British Medical Journal*, 1888.)—A man, aged fifty years, fell from a cart four days before. Insensibility present, which as far as could be ascertained came on immediately; continued upon examination. After four days stupor existed. There were found left brachial motor monoplegia and partial paralysis of the left facial nerve. Barely perceptible insufficiency of the left leg existed. There was a bruise over the upper part of the right fissure of Rolando. This condition of things persisted, and the ninth day the patient became comatose. Swallowed very poorly. Respiration became stertorous, 12, 14, 6 a minute.

Trephined over the bruise on the scalp. A clot was found at the anterior and inferior edge of the trephine-opening. A second opening was made immediately below and in front of the first and a thick clot removed. There was immediate relief, he asking for water, and swallowed it. He moved the left arm and leg while on the operating-table. Complete recovery.

CASE VIII. (Fröhlieh, *Münch. med. Wochenschr.*, 1888.)—A boy, aged seventeen years, received a blow on the left parietal region and walked to his home, some distance. In an hour and a half pain and delirium appeared, with vomiting. Stupor was present. He was found by his physician in a dying condition five hours afterward.

Autopsy. A bruise in the left parietal region. No fracture of the skull. Extra-dural hæmatoma from the supra-orbital ridge to the coronal suture. Hemorrhage from the branch of the middle meningeal. Dura was depressed 4 cm.

CASE IX. (Briggs, *Nashville Medical Journal*, xlix. 193.)—A man, aged fifty-one years, had received several blows from the handle of a whip, but walked home. A scalp-wound was found in the right temporo-parietal region. Complete coma in eight hours. Stertorous respiration. Pulse full. Left hemiplegia. Right pupil dilated and did not respond to light. Twenty-three hours later operation.

Trephined. Star-shaped fracture found. Extra-dural clot one inch thick and covering the entire right hemisphere was found. A little bleeding from the exposed small branch of the middle meningeal. Profuse venous bleeding from

the right lateral sinuses checked by lateral pressure. Sponges held in place by pressure. Forceps left *in situ*. Patient's condition alarming. Pulse 110. Patient asked for tobacco and answered questions. Sponges removed on second day. Severe hemorrhage checked by gauze tampon. Recovery, with almost complete loss of vision in the right eye and paresis in the left arm.

CASE X. (Calder, *British Medical Journal*, 1889.)—A man, aged twenty-six years, fell down stairs; was unconscious, but recovered consciousness in seven hours. Slight pain behind right ear for five days, then became unconscious again. Sensation and motion of limbs normal. The right pupil a little larger than the left; both reacted. Ptosis of the right eyelid and strabismus of the right eye. Pulse slow, full, and regular. Just above the right ear was found a slight swelling under the skull. Pupils became dilated on sixth day. Pulse 48. Occasionally patient pointed to the swollen spot. On the seventh day unconsciousness deepened; coma at 5 P.M.

Trephined one and a half inches in front of the right ear. A clot was found forming, about an ounce of blood on the surface of the dura and adhering to it. In the evening the pulse rose to 80 and consciousness was completely restored. Ptosis and squint disappeared on the fourth day. Discharged in one month, well.

CASE XI. (Deaver, *Journal of Nervous and Mental Disease*, 1890.)—A man, aged twenty years, received a pistol-wound in the left temporal region. Conscious. No symptoms. Next day aphasia.

Trephined over the lower end of the left fissure of Rolando. Bullet imbedded in the skull and fracture of the inner table. No depression. Extra-dural clot. Hemorrhage from the posterior branch of the middle meningeal. Complete and quick recovery.

CASE XII. (Deaver, *Journal of Nervous and Mental Disease*, 1890.)—A man, aged thirty-four years, fell twenty feet down a hatchway. A scalp-wound over the right parietal eminence. Respiration 14, temperature 97°. Conscious, but a little dull. In an hour and a half restlessness, twitching of the right leg, arm, and face, respiration labored, then in half an hour convulsions of the right side, becoming rapidly general.

Trephined four hours later under the wound, but no fracture was found. Four ounces of extra-dural clot. Death from shock.

Autopsy. A fissured fracture of the left parietal bone, starting immediately below the trephine-opening and extending to the base of the skull.

CASE XIII. (Deaver, *Journal of Nervous and Mental Disease*, 1890.)—A man, aged twenty-three years, fell fifteen feet, striking on his head. Complete unconsciousness. Pupils reacted, respiration 24, and pulse 100. Scalp boggy over the whole of the left side and vertex. Partial consciousness returned in twenty minutes. Epileptiform convulsions, breathing embarrassed, pulse full and strong. Cyanosis. Respiration slow, deep, and stertorous. Pulse slow and full. Right side slightly spastic, left side paretic.

On raising the scalp there was found a fissured fracture on the left side following the coronal suture. Trephined over the lower end of the fissure of Rolando. Anterior branch of the middle meningeal ligated. Bleeding continued, and a ligature was placed on the posterior branch of the middle meningeal. Four hours after partly rational. Six hours after, pulse 96, respiration 28. Recovery complete. Two slight convulsions on the second day.

CASE XIV. (Ransohoff, *Annals of Surgery*, 1890.)—A man, aged twenty-eight years, a merchant, fell eight feet from a ladder. No unconsciousness and no symptoms. He went about his business. Eight days later headache and unconsciousness. In three hours stertorous respiration, pulse 40 and full. Coma. No conjunctival reflex. Twitching movements of the face and both sides of the body. No paralysis. Right pupil contracted, sluggish. Left pupil dilated unduly and fixed. No evidence of traumatism on head. Urine, specific gravity 1040 and contained sugar. Temperature 107°. Stertor and coma. Paresis.

Trephined over the left temporal bone two inches above the external angular process, and six ounces of extra-dural clot removed through the opening, 1 x 2 inches. All hemorrhage ceased on the removal of the clot. Pulse was

140, and collapse supervened. Next day consciousness returned. Hemorrhage on the ninth day; recurred on the tenth in spite of packing, and the source could not be found.

Ligation of the carotid artery, septic course of wound, and death on the fifteenth day from hemorrhage from carotid. Urine normal twelve hours after the operation.

CASE XV. (Ghent, *Gaillard's Med. Journal*, August, 1891.)—Blow from a crowbar over the right parietal bone. Unconscious for twenty minutes, then recovered and walked 300 yards. Conscious for five hours, and then unconscious. Pupils dilated, respiration heavy and stertorous. No movements on the left side. Fifth day unconscious, pulse 46, respiration slow, involuntary micturition and defecation.

Trephined over parietal bone in the wound. No fracture or depression. One and one-half ounces of extra-dural blood-clot removed. On rousing from chloroform spoke and recognized friends. Moved the left arm and leg on the day after operation. Recovery complete, though delayed by suppuration.

CASE XVI. (Redman, *Medical News*, 1891.)—Conscious for half an hour, climbed a fence and walked home, fifty yards. One and one-half hours later unconscious; pupils widely dilated, no reaction; pulse 120, wiry; respiration labored and becoming stertorous. Vomiting every few minutes. Involuntary micturition. Head held slightly to the left side. Twitching of the right arm and leg. Rhythmic clonic spasms of right arm. Scalp-wound on the right side, horseshoe-shaped and four inches long, its centre being about a quarter of an inch above the right eye. A fissured fracture of the frontal bone one and one-half inches long.

Trephined on the right side three hours after the injury; the inner table was found depressed. An extra-dural clot three inches in diameter removed. Patient could talk on coming out of chloroform. Catgut drainage. Rapid and complete recovery.

CASE XVII. (Stewart, *Edinburgh Medical Journal*, December, 1891.)—Fell into the water, striking his head on a pile. Walked toward home, and in twenty minutes fell unconscious in a convulsion. Spasms began on the left side of the body, and appeared later on the right side. A scalp-wound one and a half inches above the left mastoid process. Bone not fractured; breathing oppressed; pulse about 60, and slightly irregular; bleeding from the right nostril.

Trephined one and a half inches behind the external angular process of the zygoma on the right side. From four to six ounces of blood were removed. Dura mater flapped with each inspiration. Next morning the patient wanted to go to work. Recovery complete in three months. Interrupted in three weeks by a rise of temperature to 103.6°.

CASE XVIII. (McCosh, *New York Medical Record*, 1892.)—A boy, aged seven years, fell down stairs. Semi-comatose for twenty-four hours. On the third day stupor lessened. Legs drawn up; aphasia. On the sixth day paresis of the left upper extremity. On the seventh day paresis of the left leg and face. Convulsive movements on the left side, and finally complete hemiplegia.

On the eighth day trephined just in front of the right fissure of Rolando. Extra-dural clot found over the centre of the Rolandic fissure. Dura torn. Moved leg and arm on coming out of chloroform. Recovery complete, without convulsions.

CASE XIX. (J. W. Elliott, *International Medical Magazine*, 1893.)—A boy, sixteen years old, fell twelve feet. A small scalp-wound existed near the right parietal eminence. Stunned; regained partial consciousness; could be roused. Pulse 68, strong; respiration 20, and quiet. Hemorrhage from right ear. No paralysis, but slower response on the left side. Knee-jerk greater on the left side. In about two hours could not be roused. Pupils dilated, right more than left, and neither responded. Pulse 42; stertor, soon becoming coma. Respiration 28; pulse 40. The right pupil unduly dilated.

Operation. No depression. Trephined one-half inch above the zygoma over point of the right middle meningeal artery. Found a large extra-dural

hæmatoma. Branch of the anterior division of the middle meningeal torn and bleeding. Opening enlarged upward and backward. Brain depressed one inch. Considerable hemorrhage from the dura checked by gauze tampon. Pulse 100; respiration slow and stertorous. Consciousness returned on the second day. Recovery complete in three months.

CASE XX. (Elliot, *Massachusetts General Hospital Records*, vol. cclxxvii. p. 82.)—A man, aged twenty-seven years, fell from a freight-car. Nine hours later semi-conscious; pulse 100, and weak; pupils were equal and reacted. Œdema over the right temporal region and a small wound of the occipital region. Twenty-four hours later unconsciousness complete; no movement of the legs. Fracture of the spine in the lower dorsal region discovered. Pulse 88. Œdema of scalp increased. In forty-eight hours cyanotic. Scalp tense and very œdematous.

Trephined over the upper part of the right parietal bone, and a fracture found. Fissure running forward and backward continuous with the coronal suture, which was split open. About three drachms of extra-dural clot and hemorrhage from the longitudinal sinus. Pulse rose to 150 and the cyanosis disappeared. Answered questions the evening of the operation. In twenty-four hours right facial paralysis. In forty-eight hours restless and delirious. Death on the fifth day.

CASE XXI. (Elliot, *Massachusetts General Hospital Records*, vol. cclxxvii. p. 69.)—A man, aged forty years, fell from a freight-car and walked a short distance. Partially conscious. Pulse 48 to 60, tense and full. Left pupil dilated and reacted slightly; right pupil normal. No paralysis. Stellate wound before and behind left parietal eminence. Slight bleeding from the left ear. Occasional twitching of the right arm and shoulder. Respiration 24, natural. Wound enlarged and no fracture found. Pulse remained about 64. In the evening grew slightly more unconscious.

Trephined six hours later in the centre of the wound. Dura was tense, bulging, and non-pulsating. Dura was incised. A slight amount of clotted blood oozed out, and the lacerated brain protruded. No fracture was found. Symptoms seemed due to laceration of the brain-tissue. Dressed, with drainage. Pulse rose to 100, and varied between 70 and 100 during the night; breathed with regularity. Seven hours after operation twitching of the left arm and shoulder. Twelve hours later the right pupil was unduly dilated and convulsions were present. Cyanosis, stertor, rapid respiration, rising pulse, and death sixteen hours later.

Autopsy. Fissured fracture starting just below trephine-opening downward and forward across parietal and squamous portion of the temporal bone just in front of the external auditory meatus, lacerating the middle meningeal artery. A blood-clot, about five ounces, lying partly in the middle and partly in the posterior fossa of the skull depressed the dura about an inch. The dura was intact.

CASE XXII. (C. B. Porter, *Massachusetts General Hospital Records*, vol. cclxxvii. p. 10.)—A man, aged thirty-three years, fell down an elevator-well two flights, and was immediately unconscious. In half an hour stertorous breathing was present, but he could be partially roused. Pupils were equal and reacted alike. A slight scalp-wound was found just to the right of the sagittal suture on the biauricular line. Six hours later the left pupil was contracted and fixed, and the right was dilated and fixed. Paralysis of the left side. Complete unconsciousness. Pulse rose to 180. Operation nine hours later.

On clearing the skull, a crack was seen running downward and toward the lambdoid suture, which was somewhat separated. The superior longitudinal sinus was found to have been torn. Sutures were passed beneath the sinuses with curved needles, and packing with gauze controlled the hemorrhage. Death on the table. Dura was found torn, and the brain was lacerated extensively.

CASE XXIII. (Scudder, *Massachusetts General Hospital Records*, vol. cclxxv. p. 265.)—A man, aged thirty-two years, fell from a freight-car. Unconscious. No interval of consciousness. Pulse 64, high tension; respiration 32, and stertorous. Pupils equal but contracted, and did not react. Œdema of right

temporal region. Right eye bulged slightly, and the right upper eyelid was ecchymosed. No fracture of the skull detected. Moved arms and groaned when temporal region was pressed upon, and showed after two hours partial return to consciousness.

Operation in the right temporal region, and a fissured fracture found running to the base of the temporal fossa parallel to and just one inch behind the coronal suture. A slight depression on the anterior edge just behind and above the external angular process.

Trephined just in front of the fissure. Dura non-pulsating, dark in color, and rather tense. A little extra-dural blood removed (three or four drachms). Hemorrhage from the middle branch of the meningeal found by enlarging the opening downward and backward and the artery tied. The dura was torn along the lower portion of the fissure. A little lacerated brain-substance escaped. Gauze drain. Pulse fell during the night to 114. Roused by pressure on the third day. Conscious on the third day. Recovery complete in three months.

CASE XXIV. (*Massachusetts General Hospital Records*, vol. cclxxi. p. 186.)—Male. Fell from a freight-car. Bruise on the right side of the head. Pupils contracted and reacted but slowly. Pulse 84. In four hours convulsive movements of right face, passing to the left side of the body. Six hours later the pulse grew more rapid (160), short and weak. Stertorous respiration. Movements of the left arm after twenty-one hours.

Trephined over the left fissure of Rolando. Hæmatoma, intra- and extra-dural. Brain lacerated. Fissure of the skull directly across the vault. Death in two hours.

CASE XXV. (Park, *Medical News*, Philadelphia, December 3, 1892.)—A boy, aged fifteen years, was struck on the right side of the head by a wagon. Bruise and ecchymosis. No wound. Six days later there were no motor symptoms, but he was feverish and restless, with a rising temperature. Flattened and depressed area of skull felt back of the right parietal eminence. Linear V-shaped fissure with depression found above the right ear.

Trephined 5 cm. above the right ear, and found an extra-dural clot beginning to be organized, 10 by 6 cm. and 1½ cm. deep at the deepest portion. Recovery.

CASE XXVI. (Packard, *Medical News*, Philadelphia, 1894, p. 45.)—A man, aged twenty-seven years, fell from a wagon. Points of ecchymosis over the right parietal bone one inch from the median line. Œdema and tenderness on pressure. Patient dull and stupid.

Exploratory trephine over the right parietal bone under tender area. One ounce of extra-dural clot. No symptoms. Recovery.

CASE XXVII. (Harlam, London *Lancet*, February 24, 1894.)—A left-handed boy, aged sixteen years, received a blow above the right ear with a cricket-ball. He was dizzy, but continued to play for a while, and walked home, a few hundred yards. The accident was at 5 P.M., and he got home about 6 P.M. He soon felt sick and became semi-comatose. At 8 P.M. unconsciousness, stertor, pupils equal and reacting. Pulse slow and full; temperature subnormal. No paralysis. Three days later paralysis began in the left leg and spread to left upper extremity and side of face. Four days later more marked paralysis and reflexes absent. Anæsthesia over the paralyzed area. Pulse slower. Five days later unconsciousness less marked, but paralysis the same. Scalp showed slight abrasion at the top of the right ear.

Trephined one and a quarter inches above the right auricle. No fracture or clot. Second opening above and slightly in front of the first. A little blood from the upper posterior side. Third opening above and behind second. No clot. Hemorrhage from smaller branches of the posterior division of the middle meningeal diagnosticated. Thought best not to search further, but rely on having relieved the pressure. No drainage. In examining the third button of bone a fissured fracture of the internal table was found. Convalescence was slow, interrupted by the formation of a hæmatoma. Complete recovery in five months.

GROUP OF SUB-DURAL HEMORRHAGE CASES.

CASE I. (Schneider, *Archiv für klin. Chir.*, 1887.)—Stab-wound in the left temporal region received during a fight. Lay unconscious for an hour, as if in a swoon; then recovered consciousness, but could not speak. Could hardly move his right arm, and his right leg moved but little. Four days later right facial paralysis; consciousness complete; answered questions correctly by signs with the left hand. Paralysis of the right arm and paresis of the right leg. Pupils normal. Pulse 60. Wound nearly healed. Paralysis of the right side increased, so that finally the right arm and leg were almost powerless. Temperature remained normal. Pulse was slow; consciousness clear.

On the ninth day trephined over Broca's convolution. Dura was non-pulsating, a bluish blood-clot showing through dura. On opening the dura and removing a small clot a moderate-sized artery spurted from the depth of the wound, which was tied with catgut. The pulse rose to 80 after the operation, and speech began to return on the third day. Moved the right arm on second day. The facial paralysis had not entirely disappeared in six months.

CASE II. (Ceci, *Deutsch. med. Zeitung*, 1887.)—A fall, and two months later paraplegia followed by left hemiplegia. Ten days later the tongue turned to the right. Thickness of speech. Involuntary micturition. Coma.

Trephined over the right fissure of Rolando. Sub-dural clot found. Gradual and complete recovery. The diagnosis of a cerebral abscess was made before the operation.

CASE III. (Armstrong, *Journ. Amer. Med. Assoc.*, 1887.)—A man, aged fifty-three years, received a blow on the left forehead with a brick. Wound of scalp. Three months later paresis of the right arm and leg. Chills and fever.

Trephined over the left frontal bone. Dura dark and non-pulsating. The incision of the dura allowed the escape of fluid blood. Motion of arm and leg returned the same day. Complete recovery.

CASE IV. (Macewen, *Lancet*, August 11, 1888.)—A boy fell, bruising the face. Developed stupidity. On the sixth day convulsions began on the left side of the face, and extended to the left arm and leg. Consciousness continued. On the seventh day convulsions persisted and were accompanied by loss of consciousness.

Trephined over the centre of the right Rolandic fissure. A fissured fracture of the skull was found. The dura was dark-colored. When the dura was opened two ounces of fluid and clotted blood escaped. Recovery without incident.

CASE V. (Ball, *British Medical Journal*, 1888.)—A man, aged twenty-six years. Ten days before entrance received a blow on the head with a penknife. Since the accident there had been difficulty in speaking and pain in the head. Cicatrix small and extending deeply through the left temporal muscle, but fully healed.

Examination found motor aphasia, word-blindness, and word-deafness, but no paralysis. Five days after entrance symptoms increased, and trephining was done. A penknife-wound was found through the left temporal bone, and the dura and brain found to have been penetrated. The wound in the dura was enlarged, in which process the posterior branch of the middle meningeal artery was cut. On dilating the sinus in the brain, dark-colored blood-clot escaped. More blood-clot was washed out by irrigation through a drainage-tube. Complete recovery interrupted by the recurrence of aphasia on the third day, due to the plugging of the drainage-tube.

CASE VI. (Walker, *Medical Age*, 1888.)—A boy, aged eleven years, fell twenty feet, striking on his head. Unconscious for half an hour. Vomited frequently small quantities of blood. The next day appeared aphasia and occasional delirium. Two days later spasms of the left arm, with turning of the eyes to the left. Started when spoken to and tried to get out of bed. Third day hemiplegia present. Vision impaired. Aphasia complete.

Trephined over right parietal eminence. A fracture was found extending

from just above the external auditory meatus to the parietal eminence. There was also found a transverse fracture through the middle of the squamous portion of the temporal bone. The depressed portion was elevated. Dura was found dark-colored. Considerable hemorrhage. Clot was removed by irrigation. Motion of the arm and leg began to return in two days. Recovery in three weeks.

CASE VII. (Owen, *British Medical Journal*, 1888.)—A boy, aged nine years, fell from a cart and was stunned. Drowsy, restless, and muttering. Five days later convulsions involving the right facial and suprahyoid muscles and extending into the right arm. He had nine of these convulsions during the day. Motor aphasia was present. On the sixth day the attack was severe, involving the right sterno-mastoid and diaphragm. On the seventh day aphasia and twitching.

Trephined over the centre of the fissure of Rolando on the left side. The dura was granular and bulging. Considerable dark blood escaped, and the edge of a solid clot was exposed in the lower portion of the opening. Another trephine was placed just below the first, and the clot was thoroughly exposed and removed. Recurrence of convulsions on the next day. On the second day the wound was opened. No clot was found under the dura, but the pia was bulging and covered with healthy granulations. The pia was opened and a little fluid blood escaped. Irrigation. Two slight convulsions the next night. No convulsions after this. Discharged well in one month.

CASE VIII. (Matas, *New Orleans Med. and Surg. Journal*, 1889.)—A boy, aged nineteen years, received a blow on the left side of the head with a heavy stick, and was knocked senseless, but recovered quickly and walked home. Several hours later became worse, was stupid and staggered. Two days after the injury was semi-conscious. The right arm and leg were paralyzed. There was anæsthesia of the upper and lower extremities. Temperature 99°, and pulse 78. Swelling of the scalp over left parietal region. Answered questions incoherently. On the third day became comatose.

Trephined over left parietal eminence. The bone was slightly indurated. The dura was bulging and dark in color. One drachm of blood aspirated through the dura with a hypodermic syringe, and considerable blood trickled from the holes made by the needle. Recovery in fifteen days after slight suppuration.

CASE IX. (Allingham, *Clinical Society Transactions*, pp. 22, 221.)—A man, aged forty years, fell from a tram-car. Did not remember the accident. The next day was drowsy. The following three days was drowsy and irritable. Pupils equal and reacted. No paralysis. On the sixth day more drowsy. Respiration labored and stertorous. Convulsions beginning on the left side of the face and extending to the neck, arm, and leg of the same side. Involuntary micturition. Pupils reacted slightly, and the right was a little larger than the left. No corneal reflex. Stertor increased and convulsions became more frequent.

Trephined on the right side over the fissure of Rolando. The middle meningeal artery, posterior branch, seen intact. Dura non-pulsating; a dark mass beneath it. The dura was incised and a large blood-clot removed. The clot extended as far as the finger could reach. The bleeding vessel ligated. Catgut sutures and drainage. The next day more rational. Could move the left leg, but not the arm. Recovery interrupted by retention of the wound-discharges and suppuration. Recovery complete in two months.

CASE X. (Mills, *Journ. Nervous and Mental Disease*, 1890.)—A woman, aged sixty years, received a blow on the head. Unconscious. Boggy area over the right parietal bone. The left pupil did not react. The right pupil small and fixed. Paresis of the left arm. Right arm rigid. No sensation in the lower extremities. Breathing stertorous and sometimes Cheyne-Stokes. Involuntary micturition and defecation. For two days the pulse was from 64 to 100, and the respiration 20. On the second day moved when pressure was made upon scalp. Unconsciousness. On the evening of the second day died.

Autopsy. Sub-dural clot five inches long and two and a quarter inches broad

over the upper temporal lobe, extending under the left parietal and frontal bones. Clot over the left cerebellar hemisphere.

CASE XI. (Chiene, *Trans. American Surgical Association*, 1891.)—A man, aged forty-two years, fell from a cab and was stunned, but recovered and gave name and address. Next day conscious. Was dismissed from the hospital on the fifth day, but readmitted on the sixth day. On the sixth day he lay on the left side with legs drawn up. Twitching of left side and face, jaws set, pupils equal and reacting slightly. The arms spasmodically contracted, especially the left. The right arm used more than the left. Pulse 80. On the seventh day twitching of the left face, arm, and leg. On the following two days this twitching increased in frequency and severity.

On the tenth day trephined just in front of the lower end of the right fissure of Rolando. The dura did not pulsate and was bluish and bulging. The dura was incised, and dark fluid blood escaped. The pulse rose from 60 to 80. Rigor on the evening of the thirteenth day; otherwise convalescence was uninterrupted, and he went to work in three months.

CASE XII. (Duret, *Cong. Trans. de Chir., Paris*, 1891.)—A man, age not given, fell from a cart. Temporary unconsciousness, followed by headache. On the thirteenth day aphasia. Slight right brachial paresis. Slight elevation of temperature. Sensory paresis of the right side. No apparent lesion of scalp or skull.

Trephined on the twentieth day over Broca's convolution. Sub-dural blood-clot and fluid blood. Ligation of two branches of the middle meningeal exposed by trephine and divided at operation. Spoke after two days. Aphasia gradually recovered. Died on the twenty-first day.

Autopsy. Congestion and œdema of the meninges over the whole surface of the encephalon.

CASE XIII. (Homans and Walton, *Boston Medical and Surgical Journal*, 1891.)—A man, aged twenty-eight years, fell from a carriage and was stunned, but recovered. Pupils equal, reacted. Pulse 60. Respiration normal. Hæmatoma in the right parieto-occipital region. On the third day delirium, followed on the fourth day by stupor and aphasia. Twitching of the right arm and leg, followed later by twitching on the left side. Later spasms became general and tonic in character. Partial consciousness.

Trephined over speech-centre near the lower end of the left Rolandic fissure. Dura bulging, tense and opaque. Hemorrhage found extending about in all directions. Pia mater cloudy and black just behind the fissure of Rolando. Brain considerably lacerated. Recovery in three months.

CASE XIV. (Elliot, *Massachusetts General Hospital Records*, vol. cclxxxi. p. 41.)—A man, aged forty-two years, fell twelve feet, striking an iron bar six feet from the ground. At entrance, pulse weak and 42 to the minute. Respiration stertorous. Right pupil unduly dilated. Moved the left arm and leg freely, but moved the right only occasionally.

Trephined in the right temporal region. Dura bulging, punctured. About half an ounce of blood spurted out under great pressure. Immediate relief to stertor. Pulse rose to 120. Pupils nearly normal and equal. Drainage. Nine hours later stertor, rapid pulse, cyanosis. Bronchi full of fluid. Died in fourteen hours.

CASE XV. (Beach, *Massachusetts General Hospital Records*, vol. cclxxiv. p. 43.)—A man, aged thirty-two years, fell from a car-step, striking his head. Unconsciousness was immediate and complete. Cut over the left eye. Hemorrhage from the left ear. Pupils equal and react slightly. Bruise on occiput. On the third day convulsions. Eyes turned upward and to the right. The right side of face, arm, and leg clonic spasms. Cyanosis. Convulsions frequently repeated.

Trephined over the centre for right shoulder. Dura dark, bulging and non-pulsating. On opening the dura considerable fluid and clotted blood escaped and lacerated brain-substance. Some fresh hemorrhage. At the close of operation there was stertor; the pulse feeble, from 150 to 180. On the following day the convulsions returned. On the second day convulsions, coma, and death.

CASE XVI. (A. T. Cabot, *Massachusetts General Hospital Records*, vol. cclvii. p. 214.)—Injury not known. Unconscious. Pupils equal, slightly contracted and reacting. Pulse full and slow. Respiration deep and regular. Bleeding from the right ear and left nostril. No paralysis. Œdema of the right occipital and post-parietal region, and a depression felt the size of a twenty-five cent piece. Ten hours later partially conscious. The following two days remained stupid, with conjugate deviation of the eyes to the right.

Trephined at the seat of the injury—that is, the upper posterior parietal region on the right side. Depression found less than one-eighth of an inch. Blood-clot from surface of dura. Dura non-pulsating. Four days later convulsions of the left arm, leg, and face. Dura opened, and about one and a half ounces of blood-clot removed, coming from the region of the fissure of Rolando. Dura sutured and skin wound closed. Two days later restless. On the third day spasms of the left side and face. On the fourth day restless, ptosis of the right eyelid, and dilatation of the left pupil. Convulsions of the left arm, leg, and face. Partially conscious in the afternoon. On the fourth day suddenly became comatose and died.

CASE XVII. (C. B. Porter, *Massachusetts General Hospital Records*, vol. cclix. p. 136.)—A man, aged twenty-four years, was knocked down in the street. Conscious, but dull. Pupils equal and reacting. Twenty-four hours after the injury pulse slow. Swelling in the right lower occipital region. At the end of thirty-six hours increasing stupor. During the next two days improvement in the mental condition, and wanted to leave the hospital, and was discharged against advice. Four hours later brought back restless and delirious. Convulsions of the left arm; head thrown back; right arm rigid. Respiration labored and stertorous, 30 to the minute. Right pupil dilated; left contracted and no reaction. Unconsciousness, coma; death four days after the injury.

Autopsy. Fissure in the right occipital fossa running into the foramen magnum. Small extra-dural clot in the right occipital fossa. Fissure running through the right occipital fossa across petrous portion of the temporal. Considerable sub-dural hemorrhage at the base of the left middle and anterior fossa. Laceration of brain.

CASE XVIII. (Wells, *N. Y. Medical Record*, May 14, 1892.)—Fell in the street. Unconsciousness was followed by frequent general convulsions. In three days convulsions became nearly constant. A small scar exposed on the left side of head above and in front of the ear.

Trephined under the scar and over the motor centres. Dura found adherent; no fracture, no depression. Dura dark, non-pulsating. On opening the dura a handful of clotted blood escaped, and the fissure of Rolando was exposed. Asked for a drink on coming out of ether. Recovery interrupted by occasional convulsions. Gave a history of a blow on the head three weeks before the present fall, followed only by severe headaches and weakness of the left arm and leg. Temperature 105° at the time of the operation. Blood-clot showed evidences of beginning breaking-down.

CASE XIX. (Bremer and Carson, AMERICAN JOURNAL OF THE MEDICAL SCIENCES, Phila., 1892.)—A man, aged twenty-one years, fell one story, but walked home. Temporary unconsciousness and fall one week later, then aphasia. Right facial paresis and tongue protruded to the right. Loss of sensation over the whole of the left side. Agraphia. Paresis of the right hand in about two weeks; this increased, and the right leg began to be involved. Dulness of mind.

Trephined one and a quarter inches behind the external angular process of the parietal bone, and a quarter of an inch above the base-line. Dura dark and cloudy, non-pulsating. A large amount of fluid and clotted blood under the anterior branch of the middle meningeal artery between the dura and arachnoid, which was flat and empty. On the fourth day recovered from aphasia. On the sixth day recovered from agraphia. Complete recovery in nineteen days. Hemorrhage in this case from the anterior main branch of the middle meningeal.

CASE XX. (Mynter, *Annals of Surgery*, 1894, pp. 19, 539.)—A man, aged twenty-five years, received a blow on the head with fist. Face and eyes

swollen; severe pain in the back of head. On the twelfth day clonic spasms of right side of face and arm. Paresis of right side of face. Tongue drawn to the left. Left pupil contracted. Anæsthesia of face. Paresis of right arm and leg. Aphasia complete. Temperature normal. Pulse 96. On the thirteenth day could move right arm and leg, and seemed to understand questions, and to try to answer them. Restless. Pulse 120.

Trephined over speech and face centre. Dura bulging and bluish. On opening a stream of black blood spurted several feet into the air; three ounces evacuated. Clot three inches square and one-quarter inch thick. Brain then pulsated plainly, but appeared "compressed inward" and "excavated."

Sutured dura with catgut. Button replaced. No drainage. In two days said "yes" and "no," and in four days aphasia had disappeared. Paresis well in a week. Discharged well in four weeks, except for slight weakness of right hand.

CASE XXI. (Walker, *Cincinnati Lancet-Clinic*, June 17, 1893.)—A man, aged twenty-three years, received a blow on the left side of the head with a billiard cue. On the third day delirium. Right facial paralysis. Left pupil dilated and non-reacting. Involuntary micturition and defecation. Temperature normal. Scalp-wound two inches long on the left side of the head. Linear fracture of skull found under wound. Trephined under wound and dura found dark blue and bulging. A two-ounce blood-clot removed. Facial paralysis had disappeared on recovery from ether. On the eighth day hernia cerebri appeared and gradually increased to the size of a hen's egg. Hernia sloughed off in four weeks. Recovery in eight weeks.

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II.

MIDDLE MENINGEAL HEMORRHAGE.

BY G. L. WALTON, M.D.,
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THE subject of middle meningeal hemorrhage has been exhaustively treated in the foregoing paper. There are, however, a few exceptional conditions, of importance from a diagnostic point of view, to which I would call attention.

In the first place, it should not be lost sight of that a sinus as well as an artery may be ruptured by wounds, as the following case (seen in consultation with Drs. Homan and Hildreth, fracture of the base with hemorrhage having been the probable diagnosis) illustrates:

Male, aged nineteen years, received a blow on the left jaw in a friendly sparring-bout; reeled; became unconscious in a few minutes. Within four hours tonic rigidity in all extremities had appeared, preceded by restlessness. Pupils first dilated, then contracted; the left slightly larger. Clonic spasm of right hand added to rigidity. Respiration tertorous. Ankle-clonus. No bleeding from the nose or ears, or under he conjunctiva. Eyes deviated to the right; head turned somewhat to he left. On the fourth day twitching of the right eyelid and angle of the mouth; extremities relaxed and paralyzed; slight twitching of right

hand and foot. Pulse at first 100, later 58 to 84. Temperature rose on sixth day to 106.4°, pulse to 130. Death on sixth day.

Autopsy by Dr. Swan. Surface of brain covered with extravasated blood under dura; most on left side and over occipital lobes. Middle fossa of skull contained several ounces of same, and base of brain covered with blood which extended into the vertebral canal. Small rent in left lateral sinus; no fracture.

To show that this is not a unique case, Dr. Pinkham, of Lynn, tells me that he found hemorrhage from the *longitudinal* sinus as cause of death following a blow received in a sparring contest in that city. He has not given me the details.

The next point to which I would allude is—

The hysteroid state. One factor in the diagnosis of hemorrhage, not mentioned in text-books, has impressed itself upon me, namely, the period of semi-consciousness which in certain subjects follows concussion and laceration, and gives rise to the suspicion of some more serious gross lesion, as hemorrhage. This condition is particularly likely to follow severe concussion in the case of young girls, though it also appears in adults, being by no means confined to patients previously disposed to hysteria. It seems rather the effect of profound shock and injury to brain-substance. It may, perhaps, be regarded as allied to the genuine traumatic neuro-psychoses. The patient, say a young girl, receives a severe blow on the head; a period of true unconsciousness follows. There is no bleeding from the nose or ears, no sub-conjunctival hemorrhage, no convulsive motion, no deviation of eyes or disturbance of pupils. The breathing is regular and of normal character. Notwithstanding the absence of other untoward symptoms, complete consciousness does not return for a number of days, or even weeks. There is apt to be in these cases retention of urine. After a number of days the question naturally presents itself, Have we not to do with a hemorrhage, and should not trephining be considered? The absence of all symptoms, excepting the unconsciousness, should lead to the suspicion that we have to do with a mental state, rather than with a gross lesion.

In one such case, carefully watched for days by Dr. Richardson, Dr. Mumford, and myself, the first suspicion of the true condition was aroused by the struggles of the patient against the introduction of the catheter and the involuntary flow of urine accompanying these struggles. This patient, after lying for a number of days in a condition of apparent unconsciousness, finally answered a question in a perfectly natural and petulant tone of voice, as if angry at being disturbed. Gradual recovery without unfavorable symptom followed, but it was some months before the balance was perfectly restored. In a somewhat similar case, called to my attention by the attending physician, typical hysterical hemi-anæsthesia followed a protracted period of unconsciousness which had caused great uneasiness and uncertainty of diagnosis.

Various mental conditions may follow this condition, among which refusal to talk is perhaps most frequent. This was the case in a patient in Dr. Mixter's service, seen by me in consultation. This patient, an adult, after having been struck by an engine, remained apparently unconscious for a considerable time without sign of gross lesion, with eyes sometimes held open, sometimes shut. He refused to answer questions for a long time. Whether there may or may not have been method in the subsequent mental state of this patient there can be little doubt that the trance-like condition at first observed was to be attributed to the blow.

Without referring to the medico-legal complications, which may add to the difficulties of analysis in these cases, I would simply emphasize the fact that hysteroid sub-consciousness supervening upon a blow is not to be mistaken for the deepening unconsciousness which indicates hemorrhage.

In the consideration of the collected cases the following are some of the points which have struck me as worth bearing in mind apart from the classical symptoms: Paralysis may occur without preceding convulsions. The interval of consciousness may be wanting. Rigidity may appear without preceding clonic convulsions. Bilateral rigidity does not necessarily point to hemorrhage at the base, nor even to a bilateral hemorrhage at the cortex, but may result from extensive unilateral middle meningeal hemorrhage. In the latter case the onset will be, however, unilateral.

The prognosis of aphasia, as well as of hemiplegia, resulting from cortical hemorrhage, is generally good, if the effused blood be removed by operation, even though considerable laceration has taken place.

Three months is the longest interval on record between the time of receiving the injury alleged to be the cause of the hemorrhage and the onset of symptoms resulting from hemorrhage. (I have recently seen a case with Dr. Richardson, in which five weeks of apparent health intervened, followed by coma.)

Anæsthesia may result from cortical hemorrhage, but far less frequently and less marked than motor paralysis. Stertorous respiration, though a serious symptom, does not contra-indicate operation. Coma does not contra-indicate operation. The pupils may remain unaffected.

In the exceptional event that one arm be paralyzed and the other convulsed or rigid, other symptoms must be depended on for the location of the hemorrhage. In case the injury is on one side, and the symptoms on the same side, operate on the side indicated by the symptoms, rather than at the site of the blow.

Symptoms of fracture of the base added to those of middle meningeal hemorrhage render the prognosis unfavorable. Tonic rigidity may appear in parts previously paralyzed as regards voluntary motion.

If symptoms of hemorrhage are present and operation is undertaken at all, exploration should be thorough, certainly sufficient to expose the posterior branch of the middle meningeal artery. Diagnosis of the side of lesion may be made from paralysis of the third nerve alone, if general symptoms of hemorrhage are present. In case of bilateral convulsion a dilated and fixed pupil alone may indicate the side of hemorrhage. Deviation of the eyes and head, though important in corroboration, affords little help as a localizing symptom.

III.

LOCALIZED HEMORRHAGE BENEATH THE PIA MATER OVER THE UPPER THIRD OF THE ROLANDIC AREA, DUE TO A FALL ON THE HEAD:

LOCALIZED CONVULSIONS WITHOUT LOSS OF CONSCIOUSNESS; LATER, SLIGHT MENTAL CONFUSION WITH PARALYSIS OF THE AFFECTED LIMBS, FOLLOWED BY PROLONGED COMA AND DEATH.[1]

By James J. Putnam, M.D.,
of Boston, Mass.

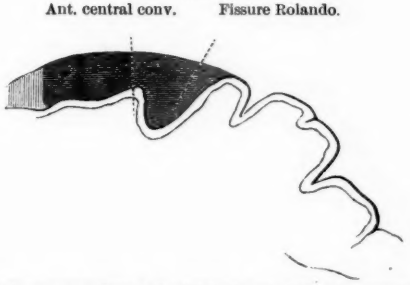
The patient was a lady, seventy-two years old and of delicate health. The accident happened in the following manner: The house in which she lived was entered by a door at the top of four stone steps. Having walked up these steps she pulled the bell. The wire broke with her weight, and she fell backward onto the sidewalk. She was not fully stunned, however, and was able with some assistance to walk into the house and up a flight of stairs to her room. Dr. C. P. Putnam was called, but found her in good condition and discovered no signs of injury beyond a bruise and swelling of the scalp at the vertex. Later, I saw her a number of times in consultation. For twenty-four hours she seemed to be doing well. Then spasms began to occur in the left arm, and to a less degree in the left leg and the left side of the face. They were slight, of jerking character, not confined to any single segment, and not attended with loss of consciousness. These spasms recurred at short intervals during about two days. As they passed away the limbs became paretic and eventually paralyzed, and at the same time the patient's mental condition became disturbed. She grew somewhat talkative, but what she said was often irrelevant, the whole condition somewhat suggesting that of a person recovering from ether. Headache was scarcely complained of at all, and the pulse and temperature were unchanged or slightly increased. At the end of about two days more this state began to pass over into one of coma, which gradually

[1] Reported in connection with Dr. Scudder's paper.

deepened until her death, which occurred a few days later, or about ten days from the date of the accident. At no time did the pulse show the slowing usually supposed to attend compression. There was very little change in temperature at any period, but toward the day of her death it gradually rose to a slight degree.

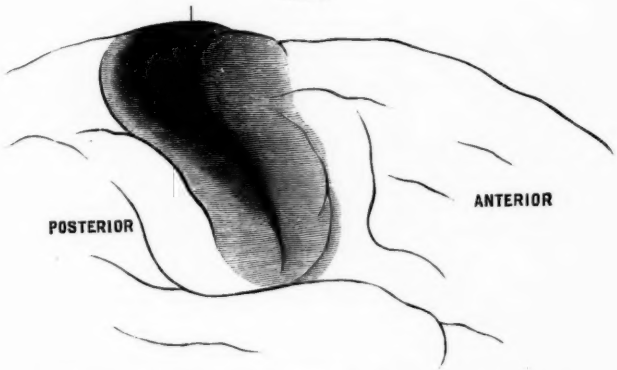
The post-mortem was made by Dr. W. F. Whitney, twenty-one hours after death, in the presence of Dr. C. P. Putnam and myself, and the following notes with the accompanying sketches were made by me on the spot.

FIG. 4.



Represents a cross-section at the thickest part of the clot, parallel to the median fissure and close beside it.

FIG. 5.



Shows the tapering of the clot toward its lower end, and indicates that the neighboring convolutions were covered with a thin layer of blood.

There was an extravasation of blood beneath the scalp covering the vertex and upper part of the occipital bone. The bone was not fractured. An extensive subdural hemorrhage covered the right hemisphere of the brain over its posterior half to near the point of the occipital portion. Besides this, a thick clot lay in the fissure of Rolando, at its upper portion, with the pia stretched smoothly over it. The central convolutions were forced apart by the clot at their upper end, and the

anterior central especially had evidently been subjected to considerable pressure. The arteries at the base were highly atheromatous.

The chief interest of this observation lies in the fact that the clinical features were somewhat different from those ordinarily seen in cases of hemorrhages of localized character, and in the indication furnished by the post-mortem, that an operation might perhaps have been done with benefit. It is almost needless to say that the propriety of operating was repeatedly discussed by my brother and myself as the different phases of the illness showed themselves, and had the signs of compression been more clear, or had the patient been younger and stronger, operation would have been decided on. But the earlier symptoms seemed to point to laceration of the cortex more than to localized hemorrhage from the middle meningeal artery, and the later symptoms were of equivocal significance. The idea of a defined clot from the pial veins did not occur to us, but we did discuss the probability of extensive and spreading sub-dural hemorrhage, and decided against an operation to relieve it in view of the uncertainty of the diagnosis, the patient's age and naturally feeble condition, and the prostration induced by the accident. I do not even now think that we were wrong, and it is doubtful whether the recovery would have been complete at the time when it would have seemed justified by the urgency of the symptoms, even if the operation had been primarily successful.

With a younger patient the results would no doubt have been excellent.

The history of this case, as of so many others, shows the unreliability of the absence of "pressure-pulse" as disproving compression.

Symptoms of cerebral excitement have been observed in cases of hemorrhage involving the cortex, and are especially noted by Gowers in his *Diseases of the Nervous System*. In uncomplicated middle meningeal hemorrhage such symptoms must be rare, if they are ever observed, unless the dura is broken through. The occurrence of such an isolated, defined, and large pial hemorrhage as this must be very rare, and, as the case puzzled us, the report of it may help to solve a doubt for someone else. [Since this report was first made microscope sections have been prepared from the dura with its hemorrhage, and the existence of an old and vascular false membrane established. It was from these vessels that the sub-dural hemorrhage had occurred.]

A CONTRIBUTION TO THE PATHOLOGY OF LARYNGEAL PHTHISIS.

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Laryngeal phthisis is acknowledged to be rarely a primary disease, whilst, on the contrary, it is quite a common complication in pulmonary tuberculosis. Regarding its origin in the latter case, auto-infection is freely accepted by all, though there still exist those who hold that the deposits of tubercle bacilli in the laryngeal tissues have been conveyed there chiefly by the blood and lymph streams, as opposed to those who hold this to be the rarer channel of infection as compared with their introduction from the surface, being carried from the lung in the sputum.

There can be little doubt, as far as clinical and pathological evidence shows, that both these channels of infection exist, either acting separately, conjointly, or one following the other.

The shallow tubercular ulcers are the product of surface-infection, and probably that form of disease which starts between the muscular layers and deep to them is caused by the first-mentioned cause. When deep involvement and superficial ulceration are present it is difficult to form an opinion as to whether blood or lymphatic infection is present, or whether the infection is exclusively from the surface.

The points to which I wish to draw attention are chiefly that the surface-infection is caused originally by the micrococci present along with Koch's bacillus in the sputum, and that the ulceration and tubercular infection are secondary to abscess-formation in the epithelium; and incidentally to the question as to whether the bacillus itself often finds its way between the epithelial cells.

To deal with the latter question first, as the evidence is chiefly negative, I quote Fränkel,[1] being, as far as I can find, the only observer who has absolutely demonstrated Koch's bacillus in the inter-epithelial spaces. For my own part, I must say that I have failed to demonstrate tubercle bacilli in the epithelium, even when the corium has been partly destroyed by ulceration, though the greater the depth of the epithelial ulcer apparently the greater is the probability of finding them.

Heinze[2] in his observations on the cadaver most emphatically corroborates the fact that the shallow ulcers are non-tubercular, when he says that out of fifty lesions 83 per cent. were specific and 17 per cent. were not; these latter were shallow ulcers, and no bacilli were found in them.

Whether Heinze himself intends the interpretation here put to his

[1] Deutsche med. Wochenschr., 1879, No. 2.
[2] Bosworth: Diseases of Nose and Throat, pp. 608 et seq.

investigations one cannot tell, but it certainly bears that rendering well, and one is justified in suggesting it.

Basworth[1] makes various allusions and remarks on the subject, some of which are repeated here from the direct bearing they have on the subject.

He says, "The theory of Louis, ... that the laryngeal ulceration results from the eroding action of the sputum as it makes its way over the laryngeal tissues from the diseased parts below, is at the present day indorsed by but few observers;" and further on, "that auto-inoculation of the laryngeal membrane is altogether probable; that this should occur, however, it is necessary that the lesion should be the seat of catarrhal erosion." To quote once more from the same author, "the influence therefore of a chronic laryngitis as a predisposing cause of tubercular disease is altogether probably greatly over-estimated, although it is undoubtedly true that a phthisical patient suffering with chronic laryngitis, especially if this has resulted in erosions, is more likely to develop tubercular action than one in whom the laryngeal membrane is in a state of health."

Regarding the theory of Louis one only needs insert the term micro-organism-laden before sputum, and the theory is quite that usually accepted. With the second quotation not many will disagree, though for my own part I must admit that I look upon a catarrhal laryngitis in tubercular disease of the lungs as being a distinct warning to attend to the larynx.

Moritz Schmidt[2] agrees with Schottelius's view, that in hypertrophic conditions of the interarytenoid space the bacillus-laden sputum is retained between the rugæ, and by maceration and irritation causes ulceration, and thus the bacillus finds an entrance into the tissues.

Here, again, is corroborative evidence in favor of a discontinuity of surface being requisite before infection takes place. My own observations quite tally with this—that is to say, ulceration is frequently present at the base of the clefts, whether caused by surface ulceration or by microscopic abscess-formation, I cannot say; probably both, for the epithelium is very thin in this situation when compared with that over the hypertrophies.

From the foregoing *résumé* it is clear that most observers consider destruction of the epithelium one part, if not in its entirety, the first stage to tubercular ulceration in the larynx; the point which now remains to arrive at is, how this ulceration takes place, whether by desquamation or otherwise.

A by no means unimportant question here is the "catarrhal" ulcer; firstly, does it exist? One must admit the existence of ulceration, neither

[1] Die Kehlkopfschwindsucht, etc., Leipzig, 1879 (Lennox Browne).
[2] Moritz Schmidt: Die Krankheiten der oberen Luftwegen, p. 289.

tubercular nor syphilitic; but is it catarrhal? If a catarrhal ulcer exists, and comes into existence by simple erosion, always excepting those cases where excretion is pent up in a crevice, then, naturally, nothing further is to be said regarding the ulceration.

But in none of my sections has any evidence of ulceration thus produced been found, nor has it been proved elsewhere.

In many cases of hypertrophy of the posterior wall of the larynx the masses do not show changes from simple hypertrophy, though pulmonary phthisis is clearly present. The posterior wall is chiefly referred to for the reason that the curette can easily remove hypertrophic masses of tissue here, whereas in other situations the surface only of the ulcer can be scraped off.

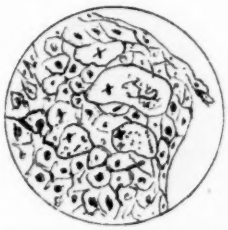
FIG. 1.

Three phagocytes in an interpapillary cleft: the phagocytes are full of cocci. × 1/12 oil immersion.

FIG. 2.

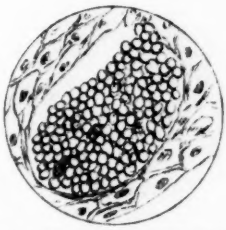
Epithelial abscess just below the surface full of detritus. × 1/12 oil immersion.

FIG. 3.



The crosses (×) are in small abscess-cavities containing cocci.

FIG. 4.



Large epithelial abscess full of pus.

In a considerable number of these little masses I have found a certain fairly uniform condition, viz., the submucous tissue is practically healthy, but in the epithelial layer there is a tendency to abscess-formation. These tiny abscesses are filled with leucocytes which have found their way up through the lower layers of epithelium, Fig. 4, and as the abscess, either by reason of normal desquamation or normal epithelial growth, approaches the surface, the leucocytes become converted into granular detritus, Figs. 2 and 3. In Fig. 1 is shown a papilla in which are seen three phagocytes conveying cocci from the surface.

In none of the epithelial abscesses have I seen a single tubercle bacillus, but I have found cocci in some. It has been shown by a large number of observers that the parts most favorable for the development of tubercular ulceration are those covered with squamous and not ciliated epithelium. This is doubtless perfectly true, but it has no actual bearing here, for, whereas it was accepted for bacilli, it must be admitted for cocci and bacilli together.

So I would put forward the following as the probable processes or stages through which the affection of the mucosa passes from health to tuberculous ulceration. Before doing so, I would say that in pulmonary tuberculosis in which there are no other micro-organisms present these arguments would not hold good.

Also, I would draw attention to the experiments which go to prove that the tubercle bacillus of Koch does not of itself cause abscess-formation, but requires the presence of cocci.[1]

Thus, as a primary factor, I would suggest that the micrococci being at rest on the epithelial surface between the attacks of coughing, and especially during sleep, find their way into the interstices between the cells. Some of these are removed by phagocytes; others are, by reason of their numbers, able to establish themselves, and, by destroying cells and phagocytes, form a small abscess. The abscess ruptures, and is still, bacteriologically speaking, non-tubercular. The ulceration continues, and as the abscess increases in depth the risk of infection by Koch's bacillus increases, and by the time submucous tissue is reached, if not earlier, this certainly occurs.

Inasmuch as these ulcerations take place very frequently in the posterior commissure, and perhaps more frequently there than elsewhere, and that they also are more frequent when hypertrophy is present, catarrh, by producing them as well as a general thickening of laryngeal epithelium, must predispose to infection.

From these and other observations it seems clear that when considering the question of treatment, whatever method is adopted, there are certain facts which might well be borne in mind, viz.:

Ulceration is not the first symptom of laryngeal invasion. The presence of other micro-organisms in the sputum probably increases the risk of laryngeal invasion.

If by intertracheal medication the bacteria can be rendered harmless, the larynx will be favorably influenced. In superficial ulceration caustic applications, viz., chromic acid 3 per cent., antiseptics and lactic acid, are all equally efficacious.

As assistant in Dr. Bond's clinic at Golden Square, I have had a very fair number of these cases, the custom being to follow Heryng's

[1] Strauss: Sem. Méd., May 10, 1894; N. Y. Med. Journ., July 7, 1894.

treatment, locally, with internal administration of creosote; but when there was nothing but simple superficial ulceration, 3 per cent. solution of chromic acid carefully applied yielded excellent results, and this is not to be wondered at, for it hardens the membrane to act as a protective investment. Thyroid extract, so beneficial in lupus, yielded no results in any cases so treated in the forementioned clinic.

RAPID GLIOSIS OF THE SPINAL CORD, WITH REPORT OF A CASE.[1]

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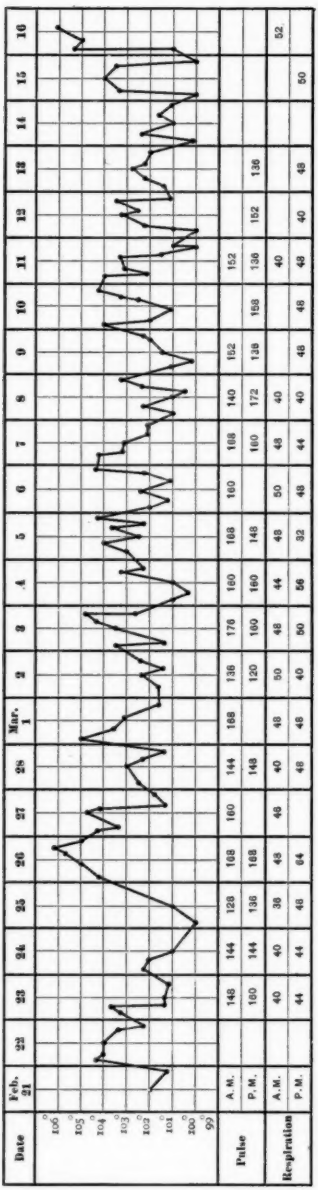
During the past ten years there has come into existence an abundant literature illustrative of the clinical and pathological features of that disease of the spinal cord of which the essential feature is a widespread neurogliar hyperplasia, and to which the name syringomyelia has been very generally misapplied, owing to the fact that cavity-formation is a frequent (though unessential) accompaniment of the process. A characteristic of a very large majority of cases of this disease is a chronic afebrile course. Occasionally, however, it happens that a pathological process, consisting of neurogliar hyperplasia of the spinal cord runs a rapid febrile course, and presents to the clinician a picture which has few points of resemblance to the typical chronic cases. The infrequent occurrence of these cases, the difficulties that attend their recognition during life, and their pathological characteristics render it important that they should be put on record.

R. R., aged one year, was admitted to the Babies' Hospital on February 21, 1894. The family history is good. There are two other children by the same parents who are healthy. About three months ago the child, Robert, was noticed to be weak in the right arm and hand, and it was observed at about the same time that the head dropped forward. The motor paralysis of the right arm gradually increased. The child had been fed on milk and oatmeal or other cereals.

On examination the child was found to be well nourished. There was complete loss of power in both arms, including loss of power in the shoulder-muscles. The muscles of the arm and hand were all markedly atrophied and flabby in consistence. The muscles of the neck were

[1] Read before the New York Neurological Society, by Dr. Herter, December, 1894.



exceedingly rigid, and the head was carried stiffly, slightly in advance of the normal position, with the face looking directly forward. Lateral and rotatory movements and extension of the head were very limited. There seemed to be some loss of power in the legs, but it was difficult to be sure of this. The knee-jerks were greatly exaggerated, that of the right side being especially active. Ankle-clonus was obtainable on both sides, but was better developed on the right than on the left side. The abdominal and epigastric reflexes could not be elicited. In the pharynx there was a very marked bony prominence, corresponding to the level of the third cervical vertebra. The eight upper incisors were slightly decayed and very short. The lower incisors were notched. There was considerable dyspnœa. Respiration was rude over almost the entire chest. There were medium or fine râles over the base of the left lung. The temperature on admission was 102°. On February 22d the temperature reached 104.5°, and the patient was restless during the night. On February 24th the temperature fell from 102° to 100°. There was repeated vomiting and breathing was labored. On February 26th the temperature rose to above 106°, and fell on February 27th to 103.5°, from which it again rose nearly to 105°, to fall later on the same day to 101°. The patient vomited twice on this day. On February 28th the breathing became entirely thoracic. Vomiting occurred at 8 A.M. and at 1 P.M. On March 1st the temperature reached 105° and fell to 101.5° later in the day. On March 2d the patient refused the morning feedings and vomited at 4 and at 7 P.M. On March 4th it was noted that there was general partial analgesia below the arms and that power in the legs was not essentially changed. The pupils responded to light, were equal, and were normal in size.

The breathing still continued thoracic. Vomiting occurred at 4 and at 7 P.M. On March 5th vomiting occurred at 3 and at 7 P.M. On March 6th vomiting occurred at 5 P.M. The patient was very restless during the night and vomited once. On March 14th there was an abundant purulent discharge from the left ear. Throughout the right lung were many fine, moist, crackling râles. There were no signs of consolidation. There were coarse râles over lung at the base behind. Vomiting occurred at 12.30 P.M. There was considerable nasal discharge. The condition was stationary as regards other symptoms. On March 16th, at 8 A.M., the face was flushed, there was slight strabismus, and the head was rolled continually from side to side. The pulse was hardly perceptible, and at times there was marked cyanosis. The temperature rose to 106° and the respirations to 52. At about 6 P.M. the patient died.

During the time that the patient was under observation the temperature was extremely irregular and frequently very high. Thus, on February 21st it rose to 104.5°, on February 26th to 106°, on February 27th to 104.4°, on March 1st to 105°, on March 3d to 104.8°, on March 5th to 104.5°, on March 6th to 104.5°, on March 10th to 104°, on March 11th to 104°, on March 15th to 104°, and on March 16th to 106°. The pulse varied from 120 to 172. The respiration varied from 32 to 52 per minute. The patient was fed on cream and barley water or peptonized milk, and was moderately stimulated most of the time by means of whiskey. He also received small doses of calomel from time to time. The ice-cap and alcohol sponge-bath were used for restlessness and for the reduction of temperature.

Pathological examination. The medulla, pons, crus, part of the cerebellum, and a portion of the basal ganglia on either side, the entire spinal cord, and the cauda equina were removed ten hours after death, and soon placed in Müller's fluid. The spinal cord was cut from the medulla at a point half an inch below the anterior pyramids. The surface of the lower end of the medulla presents a large, irregular, somewhat projecting grayish mass, of firmer consistence than the normal substance of the medulla. It occupies almost the entire right side of the medulla, except a small zone anteriorly, and the left side of the medulla with the exception of a narrow irregular border around the edge. The medulla is considerably enlarged from before backward and from side to side (three-quarters of an inch from side to side and five-eighths of an inch from before backward). (Figs. 1 and 2.) The medulla is almost the same size at its upper and lower ends. The spinal cord is much enlarged at its upper end, being seven-eighths of an inch in diameter from side to side, as the specimen lies on its posterior surface, and half an inch from before backward. The very considerable enlargement of the cord extends to the eighth cervical segment. From this level to the sixth dorsal segment the cord is still somewhat larger than normal, but below this point it appears of normal size. The cord measures half an inch from side to side at the level of the first dorsal segment, and three-eighths of an inch at the seventh dorsal.

The specimen was hardened in Müller's fluid and alcohol, and sections were made from each segment of the spinal cord and from the medulla and pons. The sections were stained by Weigert's method, and by Van Giesen's picro-acid-fuchsin method. In sections made at the level of the first cervical segment the nervous structures are so extensively replaced by the elements of the new growth that it is with

difficulty that the anterior and posterior parts of the cord are to be distinguished even on microscopical examination. The topography of the gray and white substances is entirely destroyed. By means of the Weigert stain, areas of degenerated nerve-fibres may be made out here and there at the periphery, especially along the antero-lateral edge of the cord, but in all other parts of the sections not even the remains of cells and fibres can be made out. The new growth has a typical gliomatous structure. It consists in some places of a delicate network of neurogliar cell-processes, with few cellular elements; in others of a more compact neurogliar network, containing a great abundance of small

FIG. 1.

Showing position of tumor in lower part of medulla.

FIG. 2.

Showing enlargement of cord and position of tumor.

spheroidal cells with small cell-bodies. The latter structures stain more deeply than the former. In most places, in addition to the small spheroidal cells, are larger spheroidal, oval, or pyramidal cells, often with several processes, and sometimes containing several nuclei. These are the glia-cells of Deiters. In some places these are abundant, in others scanty. In many places bloodvessels are numerous and large. Many of them have thicker walls than normal. Some vessels are surrounded by a moderate number of small spheroidal cells that appear like leucocytes. The central canal cannot be made out, and there are no traces of the cells lining it.

The second cervical sections differ from the first in that their topography is less confused. The remains of the anterior horns can be made out by the presence of a few shrunken ganglion cells without processes. At the posterior edge of the central canal the epithelial lining is broken through, and the lumen of the canal is filled with glia-cells of various shapes, some of them undergoing degeneration. In the vicinity of the canal are many glia-cells whose protoplasm has become granular, and which stain imperfectly. (Fig. 3.) Here and there these degenerating cell-bodies appear to have coalesced, with the formation of homogeneous, badly staining, irregular-shaped lumps. About the periphery of the cord, and almost surrounding it, is a narrow zone of partially or completely degenerated nerve-fibres. Sections from the third cervical segment do not differ materially in appearance from those of the second segment.

FIG. 3.

Second cervical segment.

Sections from the fourth segment are still greatly confused as regards the topography of the gray and white substances. The neuroglia growth continues to occupy all the cord, but there is a narrow border which surrounds the cord except at that part which lies between the posterior nerve-roots, and contains many degenerated nerve-fibres scattered among the proliferated neuroglia cells. A few fibres appear to retain their axis-cylinders, and this is apparently true also of some of the fibres in the anterior nerve-roots. The structure of the growth does not differ materially from that at a higher level, but it is noticeable that about the end of the anterior median septum lie a large number of the largest neuroglia cells, many of them undergoing degeneration. No central canal exists, but in the region where it normally is are many cells grouped closely together, amid the larger neuroglia cells, which have the character of ependymal cells with long processes. In this neighborhood are many areas of considerable size, in which the various elements of the neuroglia tissue are undergoing degenerative change. A few degenerated ganglion cells can be made out in the region where the anterior horns might be expected to lie. (Fig. 4.)

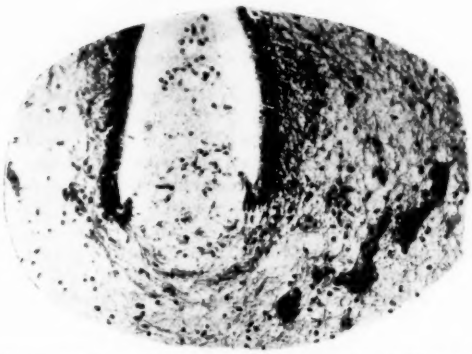
In sections from the seventh cervical segment there are clear indications of the pressure-effects of a new growth developing from the centre of the cord. The glia-invaded gray substance of the anterior horns has been pressed laterally into a long narrow band of somewhat cres-

FIG. 4.

Fifth cervical segment.

centic outline, with its convex borders directed to the edge of the cord. The degenerating ganglion cells, with their shrunken cell-bodies, lie in a long narrow line in the area just mentioned, which contains a great abundance of degenerating glia-cells of the larger size. About the edge

FIG. 5.



Seventh cervical segment. Foramen and central canal of cord invaded by gliomatous tissue.

of the cord, anteriorly and laterally, is the zone of degenerated nerve-fibres already noted in sections at higher levels. This zone is considerably wider, especially anteriorly, than it was higher up. No nerve-fibres can be seen that appear normal. The inner edge of this zone is bordered

by the outwardly displaced area of gray matter just mentioned. In the region of the central canal are some appearances of interest. The canal itself is quadrilateral in shape, and is lined with an unbroken layer of columnar ependymal cells, except at the posterior wall, which is broken through by a mass of the larger glia-cells, which partially fill the canal. (Figs. 5 and 6.) A short distance posterior to the canal lies a

FIG. 6.

Seventh cervical segment.

small ring of ependymal cells, the space between this group and the canal itself being occupied by a comparatively dense neuroglia network with numerous oval, neuclei-containing cells. The more centrally situated cells are undergoing degeneration. The pial septum of the anterior median fissure is less deep than normal, having been pushed forward by the growth of the tumor, and part of the pia has been pushed to the right, part to the left of the line of the fissure. In the triangular space thus made, the neuroglia network is particularly dense, and stains deeply with picro-acid-fuchsin. This tissue contains a large number of elongated or triangular-shaped nuclei, the appearance of which suggests that they are near derivatives of the ependymal cells.

In the first dorsal segment of the cord the outlines of the gray substance are distinct, although not so distinct as they should be. Many of the ganglion cells of the anterior horns are visible, but the greater number have lost their processes, have granular cell-bodies, and are somewhat shrunken in appearance. The gray substance contains a great number of neuroglia elements, particularly great numbers of Deiters' cells, which are undergoing hyaline degeneration. The remains of the fine network of medullated fibres can be distinguished in the Wiegert preparations. The white substance of the cord is extensively degenerated, especially in the posterior columns, where no normal fibres can be distinguished. (Fig. 7.) The most striking feature of sections at this level is the presence, about the central canal, of a great abundance of nuclei, like those of the ependymal cells, which tend to aggregate in larger

and smaller groups. This is also a very noticeable feature at the third dorsal level. The chief features of the fifth dorsal segment are the presence of a few ganglion cells with intact processes, a great number of

FIG. 7.

Fourth dorsal segment. The cavity is an artefact.

normal-looking fibres among the degenerated fibres of the white columns, and numerous large masses of hyaline substance (degenerated neuroglia cells), especially about the bloodvessels of the central region of the cord. (Fig. 8.)

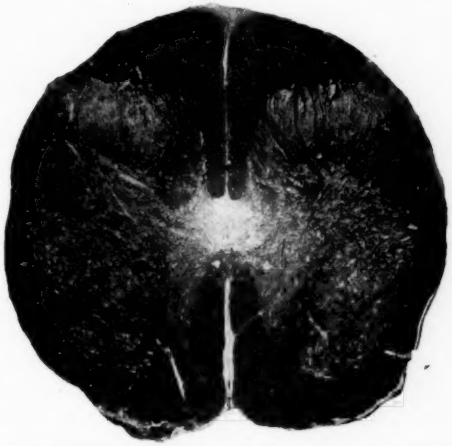
FIG. 8.

Fifth dorsal segment.

When the eleventh dorsal level is reached the white matter of the cord is quite normal even in the posterior columns, and the nerve-cells of the anterior horns and Clarke's columns are apparently intact. There continues to be, however, considerable disturbance about the central canal, where there are numbers of the nuclei above described. These, and other neuroglia cells, have invaded the anterior and posterior commissures, and are apparently responsible for the degeneration of their nerve fibres. (Fig. 9.) Throughout the rest of the cord the conditions differ little from those just described for the eleventh dorsal segment. Both the gray and the white matter are normal in the greater part of

their extent. It is only about the central canal that pathological changes are seen. The ependymal cells of the canal are surrounded, as above, by a rather dense network of neuroglia processes in which lie many

FIG. 9.



First lumbar segment. Gliomatous tissue about central canal.

nuclei having the same character as the nuclei of the ependymal cells, and numerous small spheroidal cells. The network of fine medullated nerve-fibres in this region is damaged or destroyed, especially in the parts

FIG. 10.

First sacral segment. Gliomatous tissue about central canal.

posterior to the canal. (Fig. 10.) At the level of the third sacral segment this condition is still well marked. About the area of neuroglia cell-proliferation is a narrow area of degenerative change in the gray

substance. Passing upward from the spinal cord to the medulla, it is found that the new growth rapidly decreases in size. Sections through the medulla at the level of the middle of the hypoglossal nucleus show the presence of many new glia-cells just beneath the ependyma of the ventricle. Many of the cells of the twelfth nerve-nucleus are shrunken and have lost their prolongations. The rest of the structures at this level are apparently normal, including the vessels. At the middle of the pons no deviations from the normal can be detected. Throughout the cervical and dorsal regions of the cord the vessels of the tumor are numerous, the adventitia is often thickened and is sometimes the seat of hyaline degeneration. About many vessels is a slight increase in the number of small spheroidal cells. In those portions of the cord where the new growth extends to the surface the pia shows an increase in connective-tissue cells and is somewhat thickened. The vessels of the pia are in many places apparently larger than normal, and, in some places, are the seat of degenerative change.

Outside the nervous system there are no lesions.

Having described with some detail the appearances at different levels of the cord, it remains to consider the interpretation which is to be put upon them. In structure the new growth corresponds to a glioma. It is, in fact, a glioma which involves almost the entire extent of the cord. That the growth originated in the neighborhood of the central canal is indicated by the appearances in the lower part of the cord and by the gradual advance of the tumor as one passes from below upward, from the centre to the periphery of the cord, until the entire transverse extent is implicated.

It seems probable that the new formation of glia-cells began in the proliferation of the ependymal and peri-ependymal cells. The recent researches of His and others have taught us that the neuroglia of the cord is developed from the epiblastic columnar cells, which form the foundation of and line the primitive neural canal. From this lining of germinal cells are derived the cells known as spongioblasts, which extend outward and develop into the neuroglia of the cord. By a slight modification of the germinal cells are produced the neuroblasts, which by their proliferation give rise to the nervous structures of the cord. The appearances in the preparations under consideration render it probable that the glia-cells of the new formation were derived from the proliferation of cells of the type of the ependymal cells and the isolated groups of peri-ependymal cells.

According to this view, the new cells have the same origin as the spongioblasts. There is no evidence in these preparations that there has been any proliferation of the nervous elements, the derivatives of the neuroblasts; but this may conceivably happen under some conditions. Of the two forms of tissue, spongioblastic and neuroblastic, the spongioblastic is biologically to be regarded as the lower and less differentiated form; and it is not singular that it is in tissues genetically

related to this form that the power of cell-division is retained in high degree.

It is to be regretted that the preparations under consideration were not studied according to the modern methods of cytology. There is little doubt that the adoption of the methods now in use among biologists for the study of the cell will in the future throw considerable light upon the nature of new growths generally, by making possible a more thorough study of the processes concerned with cell-division.

Regarding the relation between the position and nature of the lesion and the symptoms that were noted there are several points of interest. Although the new growth or the groups of abnormal peri-ependymal cells already referred to involved the entire extent of the spinal cord, it is only to the developed growth in the upper half of the cord that symptoms are to be attributed. The new growth reached its maximum development in the cervical region, and it is probably in the lower part of this region that active proliferation of the neuroglia cells began. This corresponds with the fact that the first observed symptom was weakness in one arm. That the right arm should have been paralyzed somewhat earlier than the left seems to correspond to the fact that at the beginning of its growth the new formation compressed and involved especially the right half of the spinal cord. The analgesia below the neck, the spasm in the leg, and the heightened reflexes are, of course, merely the usual results of a total transverse lesion of the cord in the cervical region. The spasm of the neck-muscles was the spasm characteristic of irritation of cervical nerve-roots.

The high temperature observed during the entire time that the patient was under observation cannot be referred to the pulmonary condition. It is likely that this temperature, a very unusual feature of tumor of the cord, was due to the rapid destruction of nerve-elements in the cervical cord. That rapidly destructive lesions of the lower segments of the cervical spinal cord are capable of causing a rapid, considerable, and sustained temperature is now a well-established fact. The phenomenon is observed especially in crushing-lesions of the spinal cord, in which one of us (Herter) has observed it a number of times. In rapidity of tissue-destruction the present case is, of course, not comparable to crushes of the cord, but the destructive process was sufficiently rapid to render this explanation of the pyrexia a reasonable one. The costal type of breathing which was noted was due to the implication of the phrenics by the rapid ascent of the morbid process; and it is likely that death was due, not to exhaustion, but to the implication of the respiratory nuclei in the lower medulla.

In certain respects the case here reported appears to be unique. In a fairly thorough search of the literature of new growths of the spinal

cord we have been unable to find a case with similar pathological characters in an infant of one year, where the process has been so rapid in development and has been associated with hyperpyrexia referable to this process. The case which most closely resembles ours is one reported by Miura.[1] The patient was a boy, aged eight years, who developed paraplegia, partial paralysis of the arms, and loss of sensibility below the manubrium of the sternum. There was no fever. The entire duration of the symptoms was from four to five months. Death occurred suddenly and unexpectedly. A centrally situated glioma was found extending from the upper cervical region through the lumbar region of the cord.

It is clear from a comparison with the histology of this cord that the process closely resembles the one which we have described. The distribution of the paralysis and anæsthesia and the rapid course of the disease are clinical features of both cases. On the other hand, Miura's patient was eight years of age, and is said to have had no fever during his illness.

Cases that resemble the one we have reported are described by some writers as cases of central gliomatosis. Hoffmann,[2] in his recent study of syringomyelia, insists on our recognizing two classes of cases: first, those in which there is a slow hyperplasia of the neurogliar substance, usually with eventual cavity-formation and without any enlargement of the cord or increase of consistence; secondly, cases in which there is a rapid growth of neurogliar elements with little tendency to cavity-formation and with enlargement of the cord and increase in its consistence. To the first class of cases is given the name gliosis of the spinal cord. Most cases of syringomyelia which are diagnosed during life belong in this group. The name gliomatosis of the spinal cord is given to cases of the second class on the ground that these are really cases of glioma which have extended a considerable longitudinal distance in the cord. In addition to the case of Miura already mentioned, Hoffmann places in this class the following cases:

A case of Simon's,[3] in which there was paraplegia in a girl aged twenty years, due to a large myxo-glioma with cavity.

The case of Van Giesen,[4] in which there was paraplegia of gradual onset in a farmer, aged forty-seven years, due to telangiectatic glio-sarcoma with cavity, occupying a large part of the dorsal cord.

The case of Daxenberger,[5] in which there was paraplegia in a boy, aged seventeen years, due to a glioma which extended from the upper dorsal cord into the lumbar enlargement.

[1] Ueber Gliom des Rückenmarks u. Syringomyelus. Ziegler's Beiträge zur pathol. Anatomié, Bd. xi. S. 91.

[2] Deutsche Zeitschrift für Nervenheilkunde, 3 Bd. Heft 1 and 2, 1892.

[3] Simon: Arch. für Psychiatrie, Bd. v. p. 120.

[4] Van Giesen: Report of a Case of Syringomyelia, Journal of Nervous and Mental Disease, 1889.

[5] Daxenberger: Ueber Gliombildung und Syringomyelia, Erlanger Dissertation, 1890.

The case of Seebohm,[1] in which paralysis of the legs and partial paralysis of the arms, with anæsthesia, in a man aged thirty years, were due to a spindle celled sarcoma with cavity, extending from the medulla into the lumbar enlargement.

The case of Schultze,[2] in which a Brown-Séquard paralysis with bulbar symptoms was due to a telangiectatic myxomatous glio-sarcoma extending from the medulla to the conus medullaris.

The case of Sokoloff,[3] in which a boy, aged five years, developed paralysis and atrophy in the arms and sensory disturbances in the left leg, referable to a glioma of the medulla with cavity-formation, which extended to the lower cervical region.

Although these cases, cited by Hoffmann, seem to resemble the case we have reported in their histological features, they differ from it clinically in not running such a rapid course. But, although these cases did not run an extremely rapid course, most of them died in the course of two years from the time when symptoms were first noted; and none of them lived more than three years, a common feature which is in marked contrast to the majority of cases that are described as syringomyelia. According to Hoffmann, there are pathological as well as clinical differences between cases of gliosis and gliomatosis.

Histologically, the cases of gliomatosis differ from those of gliosis in their richness in new cell-elements and their poverty in neuroglia cell-processes. Further, they differ in the greater tendency to cavity-formation which is noted in the cases of gliosis. But we cannot agree with Hoffmann that these differences necessitate the nomenclature which he employs. We believe, from study of preparations and reports of cases, that there is no essential pathological difference between the process described as gliosis and that described as gliomatosis. It is true that we may pick out cases, on the one hand, in which new cell-elements are scanty and neuroglia processes are very abundant, and in which the cord is normal in size; and, on the other hand, cases where the new cell-elements are abundant and varied, with few neuroglia fibres, and the cord is increased in size and consistence. We have, however, no intimate knowledge of the nature of these processes which justifies us in separating them on pathogenetic grounds. We do know that between the extremes of these types there are all gradations; and we have reason to believe that the process of cell-multiplication begins in both cases from the ependymal and peri-ependymal cells. On pathological grounds, therefore, we are hardly justified in making even the separation into groups which is implied by the use of the term gliosis and gliomatosis. It is proper to add, however, that Weigert and others distinguish sharply between these types on pathological grounds.

[1] Seebohm: Ueber einen Falle un Tumor der medulla spinilis Mit Syringomyelia, Stranberger Dissertation, 1888.

[2] Schultze: Arch. für Psychiatrie, Bd. viii. p. 367; also Bd. lxxxvii. p. 510, and Bd. cii. p. 435.

[3] Sokoloff: Levei Fälle von Gliom des Centralnervensystem, Arch. f. klin. Medicine, Bd. xli.

On clinical grounds a separation into groups is desirable and even necessary. The difference in progress between the extreme cases of the two types mentioned is very striking, and this entails a certain difference in the character of the symptoms; for the rapid cases are much more apt to be associated with irritative symptoms, such as pain, hyperalgesia, and rigidity, than are the cases of slow growth. In view of these facts it seems reasonable to class cases according to the rate of their progress, as cases of slow gliosis of the spinal cord on the one hand, and of rapid gliosis of the spinal cord on the other. The term gliosis is as applicable to the one set as to the other. The term gliomatosis is objectionable for the reasons above mentioned. Inasmuch as there are probably all gradations between the extremes of each type, there must necessarily be some diversity as to usage, by different persons, of the terms we have proposed. In our opinion all cases which run their course inside of two years should be classed as rapid cases. The presence or absence of a cavity is of no significance in connection with the proposed classification. It is said that cavities are less apt to occur in the rapid than in the slow cases of gliosis, and this is probably true; although it would be reasonable to expect a rapid rich-celled growth to undergo the degenerative changes which are the usual cause of the cavity-formation more regularly than a slower growth with fewer cell-elements. In our own case and in that of Miura there was no cavity-formation. It is, however, clear from the advanced degenerative changes in the neuroglia cells in certain parts of the cord which we have described that the conditions were favorable to cavity-formation, and this would probably have occurred had the degenerative process gone on a little further. Thus it may be that the rapidly fatal termination of certain cases is the chief reason why cavities are not oftener observed in them.

The majority of cases to which the word syringomyelia is applied are cases of slow gliosis of the spinal cord, and should be designated as such. If there is reason to suspect the presence of a cavity in a given case, the word syringomyelia may be added for qualification, so that the diagnosis would read, slow gliosis of the spinal cord with syringomyelia. In most cases it is impossible to say from the symptoms whether a cavity is or is not present, and the term syringomyelia has occupied far too conspicuous a place in our vocabulary.

It is to be noted that there were no secondary degenerations, ascending or descending, in the cord which we have described. At first sight this is a surprise, for the white tracts are entirely interrupted in the cervical region. But it is probable that this complete interruption was of short duration and that there was insufficient time for the development of secondary degeneration. It is remarkable

how often one fails to find secondary degeneration in the spinal cord in cases of tumor where the conditions seem adequate to cause it.

In conclusion, it is of interest to touch on some of the difficulties in the diagnosis of cases like the one we have described. The cervical paraplegia, the spasm of the muscles of the neck, the prominence of the fourth cervical vertebra led to the diagnosis of Pott's disease by several competent observers. This view of the case was strengthened by what seemed likely to be the signs of a tubercular process in the lungs and by the presence of fever. It soon became clear, however, that the fever was too high to be accounted for by the evidences of disease in the chest, and that it was probably the result of rapid damage to the cervical cord. The prominence of the fourth cervical vertebra did not appear to be associated with any abnormality of the bone, and was a misleading factor in the diagnosis.

It must be owned that the diagnosis was a difficult one to make, and we are unable at present to make a satisfactory statement of the features in which the diagnosis of such cases as these from Pott's can be made with any degree of confidence. Both Pott's and gliosis of the cord are so rare at this time of life that we have not as yet the material for formulating such points of clinical difference as may exist.

THE PHYSICAL SIGNS OF CELLULAR ŒDEMA OF THE LUNG, CONSIDERED IN THEIR RELATION TO THE PATHOLOGICAL CHANGES.[1]

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In the year 1886 I drew the attention of the Medico-Chirurgical Society of St. Louis to a form of disease of the upper air-passages and the lung that developed a history and symptoms entirely foreign to those forms of disease known in that locality. The typical form of throat disease was a solid œdema of the mucous membrane, and the pulmonary affection showed varying degrees of consolidation or infiltration of the lungs.

In the pulmonary form of the disease the mortality was very great, far exceeding the ordinary forms of pneumonia. The symptomatic history of the disease was an unusual one, and the combination of the physical signs in the different cases was at once contradictory and

[1] Read before the Eleventh Annual Meeting of the American Climatological Society.

perplexing, resembling, in certain cases, a croupous pneumonia, in others a bronchial pneumonia or bronchitis. Still the symptoms and the physical signs were at variance with those which are usually recognized as peculiar to these conditions.

In 1887 I began the study of the post-mortem appearance of these lungs, and published a report of the throat cases in a paper read before the American Laryngological Society, in June, 1889 (*New York Medical Journal*, August 16, 1889); also, one on the lung affection in March, 1890 (AMERICAN JOURNAL OF THE MEDICAL SCIENCES).

In this paper I drew attention to the peculiar cell-infiltration of the lungs, and claimed that this condition was the local effect of a general septic infection of the system. As the symptoms of the disease bear a striking resemblance to the symptoms of the influenza as described by Graves, or la grippe, of Valliex, I surmised that these might be cases of influenza. The history of 1890, 1891, and 1892, with the prevailing epidemic, proved the correctness of this view, and the cases may now be recognized simply as sporadic cases of influenza, which preceded the epidemic by several years, in the same way that sporadic cases have followed it, to the present time. My studies of this disease have been chiefly clinical, aided by the macroscopic appearance in the dead-house and microscopic sections. The limited bacteriological study that has been made for me has shown the streptococcus to be the prevailing micro-organism.

Cellular œdema of the lung has been generally called in this country the grippe pneumonia. I would consider the name "pneumonia" as improper, as only a certain number of the cases bear a resemblance to a bronchial pneumonia, and the existence of inflammatory action is very uncertain. I would consider it to be essentially an œdema of the lung in which the cell-elements, largely leucocytes, are poured out very quickly, in greater or less numbers, from the lymph and bloodvessels, producing a partial or complete infiltration of the lung. The fever and other symptoms which occur in the disease I believe to be strictly dependent upon the sepsis, and the symptoms and the amount of the infiltration of the lung will be determined by the intensity of the streptococcus poisoning. That this condition of the lung is only the local expression of a general systemic disturbance will be seen from the changes in the other organs. In fatal cases there is some change in the liver; it is usually enlarged and softened: the spleen is pulpy, giving the appearance of a septic spleen; and the heart-muscles are in many cases so softened that they can be readily torn: the kidneys are generally hyperæmic.

This conclusion is the result of a study of thirty-four post-mortem examinations made between the years 1886 and 1894. In twenty-one of these the typical appearance of the lung was alone present. In

thirteen it was complicated with other conditions. On the post-mortem table the lung, in a condition of cellular œdema, presents a swollen, congested appearance. It is generally brown-black in color, and interspersed over the lungs are areas of a lighter brown or gray. These areas may be of limited extent or they may include the greater part of the lobe. The boundaries are irregular and they are scattered over different portions of the lung. If we take up a portion which is brown black in appearance, we will find that on pressure it crepitates with difficulty. The crepitation is apparent, but the "feel" of the lung presents a degree of solidity foreign to healthy lung tissue, and the crepitation is produced only with more pressure. When placed in water it quickly rises to the surface. The cut surface is smooth and moist. If this section of the lung is placed under the microscope, it will be seen that the alveolar walls are infiltrated with numerous cell-elements, but the interior of the air-vesicles remains free. The physical signs of a lung in this condition are such that they can be fully explained by the pathological conditions. To the inspiratory sound is wanting the free flowing breath-sound of normal lung expansion. The lungs seem to be extended with decided effort, and the inspiratory sound is delayed and prolonged. In quality the sound may be slightly harsher than the pure vesicular breathing. No change, or only a slight lengthening of the expiratory sound, is apparent. The percussion-sound may be so slightly changed as to appear normal, or show only a slight shortening of the normal resonance.

Another area of the lung presents a dark-brown color. To the hand this gives a feeling of solidity. When cut it appears as a solid substance, like a piece of beef. There are no crepitations on pressure, but when placed in water it floats on the surface. Examined under the microscope we find the alveolar walls densely infiltrated with cell-elements, with a partial filling of the vesicles. Some of the vesicles are more filled than others, but in the greater part we recognize the existence of air-spaces. The amount of infiltration varies in different sections and portions of the sections, but the presence of a large number of partially filled vesicles may be recognized in all. In a cut section of the bronchus the walls may be seen thickened by the same cell-elements.

The physical signs produced by this condition of the lung depend largely upon the amount and extent of the infiltration of the bronchial mucous membrane, together with the partial infiltration of the vesicles. The percussion-sound is always deadened, giving to the finger an increased sense of resistance. In minor degrees of infiltration through the deadened sound a slight degree of pulmonary resistance may be appreciated. When the infiltration is greater we find the percussion-sound very dull; even there may be a perfect flatness. In some cases we find an exaggerated full percussion-sound, such as we find in emphysema, and it may be even tympanitic in character; but even if this is the

case, the sense of resistance is still always somewhat increased. It is difficult to explain this resonance of the percussion-sound under such conditions, and we can only believe that it is either due to the confined air in the vesicles, through the complete obstruction of the bronchii, or it may be the result of some gas-producing micro-organism which it has been claimed has been found in these lungs. Occasionally we find an interchangeability between the dull percussion-sound and the exaggerated resonance. The same place may be dull to-day and to-morrow may be hyper-resonant, and this may again be replaced by dulness.

The auscultatory signs of this degree of cellular œdema are very perplexing and contradictory. In cases where the œdema of the bronchial mucous membrane is sufficient to thicken the membrane, rendering it dry, we find the normal respiratory murmur changed into a harsh respiration, in which the inspiratory sound is lengthened, sharp, and of a high pitch, and it is immediately followed by a prolonged expiration of lower pitch. In some cases the expiratory sound is absent, and the prolonged harsh inspiration is alone heard. In cases where the œdema narrows the bronchii to a still greater degree we shall have the sibilant râles. They may be inspiratory or expiratory, or both. They resemble asthmatic râles in a measure, but they are shorter in time, the inspiratory râle rarely covering the whole time of the inspiratory act; a distinct interval of silence may or may not be followed by the expiratory râle. Occasionally we will have large-sized sub-crepitant râles, largely expiratory. They resemble dry crackling sounds, and are probably formed by the passage of the air through the viscid secretion of the bronchii. The dry and the moist râles are not, as a rule, continuous phenomena: they vary from day to day, and may entirely disappear for a time, to be again heard in varying degrees of intensity.

Where the œdema is so great that it completely occludes the bronchii we will find a complete silence over the lung. This silence is even greater than that formed over pleuritic effusion, where more or less distinct breathing may often be heard. Vocal and pectoral fremitus is wanting. This silence of the lung may continue for an hour or for weeks. It may be broken, first, by a gush of mucous râles, which can be heard during the inspiration. These râles may again disappear, and no respiratory sound may be heard. Later, they have a tendency to become permanent, and may continue as large-sized inspiratory râles for months. In other cases we have a gradual return to normal breathing without any appreciable râles.

The portions of the lungs which are of light brown, or which may appear as steel-gray, give all the evidences of a consolidated lung. They are dense and firm. The cut surface is dry, smooth, and glistening, and, when placed in water, sinks instantly. Under the microscope we find the alveolar walls and the vesicles densely filled with the cell-

elements. The physical signs of this condition are the ordinary signs of consolidation (bronchial breathing and increased pectoral and vocal fremitus). The percussion-sound is slightly flat. Occasionally the bronchial breathing is replaced by amphoric breathing. This was true where, on the post-mortem, the apex of the lung was of a light-gray color and completely consolidated. The tendency of this consolidation is to continue for a long time. It remains after all the other portions of the lung have become normal. The disappearance of the abnormal signs and the reappearance of healthy lung action is gradual. Cellular œdema of the lung seems to present a most fertile soil for the growth and development of the tubercle bacillus, and I believe we find here the origin of a large number of our present cases of acquired tuberculosis.

Occasionally we find signs of cavities. On post-mortem examination we find the cavities to be irregular in form, with soft walls, giving the appearance as if they were worm-eaten, with an entire absence of the fibroid tissue which is usually seen about tubercular cavities.

I would describe the different degrees of infiltration as types and not as stages of the disease, for I have not found them to merge from one into the other. A type which occurs in the beginning in a certain portion of the lung continues throughout the attack. The infiltration is sudden, and resolution may occur as quickly.

NOTE.—The treatment of *cellular œdema* of the lung which has given excellent results in the hands of the writer may be thus summarized: Fifteen to twenty grain doses of benzoate of sodium with half-drachm doses of solution of acetate of ammonium should be given every two hours. In addition, twenty-five drops of the muriated tincture of iron should be taken every four hours. Whiskey or champagne may be given freely, and digitalis is almost always required through the whole course of the disease. Frequent counter-irritation over the lung is desirable.

DANGEROUS CONDENSED MILK.

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DURING the course of the past summer attention was called to the sale of cans of condensed milk, with spoiled and possibly dangerous contents, and some sensational articles were published in the newspapers. I was subsequently requested by the local and State Boards of Health to investigate the matter, specimen cans of each brand at that time in

the market being collected by inspectors and submitted to me for examination. A much more satisfactory inquiry could have been made had the inspectors ransacked the retail grocery stores and collected cans showing age or distention; but all the specimens which came to me were outwardly in good condition. I had no means of ascertaining their age.

The contents of the cans in most cases had the ordinary smell of condensed milk, and were neutral or nearly so to test-paper; others were faintly acid, and one strongly so. The last had thickened into a mass with the smell, taste, and consistency of some variety of soft cheese.

I give below a table, with description of each brand, and in another table the composition. In order to find the number of times the original milk has been condensed I have divided the percentage of milk solids (*i. e.*, the total solids less the added cane-sugar) by 12.5. This is done on the assumption that the average amount of solids in ordinary whole milk is represented by this figure. The quotient divided into the percentage of fat in the condensed milk gives the percentage of fat in the original milk before condensation.

TABLE I.—MILK CONDENSED WITH CANE-SUGAR.

Brand.	Color-tint.	Taste.	Odor.	Reaction.
Tip Top,	Reddish-brown.	Very sweet.	Normal.	Very slightly acid.
Eagle,	Yellowish-brown.	Sweet and pleasant.	Normal.	Nearly neutral.
Champion,	Yellowish-brown.	Sweet and dry.	Normal.	Nearly neutral.
Daisy,	Brownish-white.	Sweet and dry.	Normal.	Nearly neutral.
Full Weight,	Yellowish-brown.	Sweet.	Normal.	Neutral.
Sweet Clover,	Light yellowish-brown.	Sweet.	Normal.	Nearly neutral.
Challenge,	Dark yellowish-brown.	Sweet.	Normal.	Neutral.
Magnolia,	Nearly white.	Cheesy.	Cheesy.	Acid.
C. and S.	Yellowish-brown.	Sweet.	Normal	Neutral.
Atlas,	Yellowish-brown.	Sweet and pleasant.	Like whole milk.	Slightly acid.
Leader,	Light brownish-white.	Sweet.	Normal.	Slightly acid.
Standard,	Brownish-white.	Sweet.	Normal.	Nearly neutral.
Winner,	Reddish-brown.	Buttery.	Faint cheesy.	Nearly neutral.
Bell,	Yellowish-brown.	Sweet and pleasant.	Normal.	Nearly neutral.
Red Cross,	Brownish-white.	Sweet, unpleasant.	Unpleasant.	Slightly acid

I have taken the number 12.5 as representing the average total solids, because the State of New Jersey makes the legal standard 12 per cent., while other States enact 13. The last number represents very nearly the true average, but at the same time so much of the milk of particular cows, or particular breeds of cows, falls below this figure that I think the legal standard should properly be made 12.5 per cent.

In the process of condensation the tint of color natural to milk is, of course, somewhat intensified; but, in addition, some of the samples have quite a decided brownish tinge. This is due to a change in the fat. After extraction with ether the fat obtained in the process of analysis from condensed milk frequently has a tint of brownish-yellow, while the fat similarly obtained from ordinary milk is colorless or faint yellow.

This change is of a character unfavorable to the use of such condensed milk in infant-feeding.

TABLE II.—MILK CONDENSED WITH CANE-SUGAR.

Brand.	Percentages.								Times condensed.
	Water.	Fat.	Caseine and albumin.	Milk-sugar.	Ash.	Cane-sugar.	Milk solids.	Fat in original milk.	
Tip Top	26.00	10.17	9.22	11.37	1.99	41.25	32.75	3.88	2.62
Eagle	28.32	9.11	9.06	10.11	1.91	41.49	30.19	3.78	2.41
Champion	27.72	9.60	9.52	10.11	1.83	41.22	31.06	3.87	2.48
Daisy	25.53	9.41	8.71	13.43	1.83	41.09	33.38	3.52	2.67
Full Weight	28.18	9.00	7.87	11.70	1.88	41.37	30.45	3.70	2.43
Sweet Clover	28.70	10.30	8.47	11.31	1.83	39.39	31.91	4.17	2.47
Challenge	28.44	9.86	8.99	10.05	1.82	40.84	30.72	4.04	2.45
Magnolia	30.04	9.48	8.17	12.81	1.91	37.59	32.37	3.66	2.59
C. and S.	26.99	9.11	8.68	16.98	2.15	36.09	36.92	3.09	2.95
Atlas	28.30	7.64	10.91	10.79	1.83	40.53	31.17	3.06	2.49
Leader	28.20	8.02	8.01	11.32	1.80	42.65	29.15	3.44	2.33
Standard	27.21	7.79	8.21	13.30	1.85	41.64	31.15	3.12	2.49
Winner	29.84	9.63	9.82	11.17	1.98	37.56	32.60	3.70	2.60
Bell	25.54	12.13	8.87	10.51	2.05	40.90	33.56	4.52	2.68
Red Cross	29.34	8.80	7.81	10.00	1.76	42.29	28.37	3.87	2.27
Average	27.89	8.67	8.82	11.66	1.83	40.39	31.71	3.69	2.52

On an average, 55 per cent. of the solid matters is cane-sugar. This, of course, imparts excessive sweetness, which renders infants fond of such milk and impatient when a change to an unsweetened diet is attempted. There is, in addition, a certain dry or even gritty taste in some samples. This is due to the milk-sugar, which being sparingly soluble, requiring from five to six parts of cold water to dissolve it, begins to crystallize out. It forms minute, hard, white, transparent crystals. This grittiness sometimes originates a prejudice, being attributed to some impurity, and to overcome this false notion some of the labels call attention to the gritty taste, and state that inasmuch as it is due to the presence of milk-sugar it is positive proof of the purity of the fresh milk used in manufacture. Of course, this is an overstatement, since the milk might be impure and yet all of its milk-sugar might be present.

The most important change is that which sometimes results in producing condensed milk of a slimy, cheesy, or semi-solid character. Such milk, in the course of time, may even become quite solid, or "go hard," to employ the term familiarly used in the trade; or it may undergo putrefaction, liberating gases which distend or burst the can.

These changes are due to specific bacterial ferments getting into the milk before the process of condensation. What most concerns us here are not the description and characters of these organisms, but the circumstances under which they get into the milk and the effect they produce.

Of those, for instance, which produce the slimy fermentation at least six species are known. They are not found in pure milk, and are not met with when the feed and fodder of the cattle, the barn and dairy, the milk-pails, utensils of every sort, and the toilet both of the cows and men are truly clean. The same remark applies to the entrance of all other fermentative and putrefactive bacteria.

Some cannot be avoided, but their multiplication can be minimized by careful handling of the milk prior to condensation, and particularly by its immediate cooling to a temperature as near 32° as practicable. Inspectors now forbid the marketing of milk for domestic use without such cooling immediately after milking, but the equal importance of so doing before condensation is not yet generally recognized. In the interim before cooling ptomaines in the nature of tyrotoxicon are generated, and act as more or less acute poisons.

Fortunately the interests of the public coincide with those of the manufacturers in most respects, and, apart from the propriety of its use for infant-nutrition, much can be said in praise of the great value of condensed mild as a manufactured product. It would be a great loss to the canner were he to use or purchase watered milk, since the chief expense is the evaporation of the water. And dairymen dishonest enough to water milk are also those most likely to have impure wells, improper food, and unclean surroundings. Not from added water, but only, in the case of condensed milk, from water used in washing utensils and in handling is danger to be dreaded. The evidence of the analysis shows that it is the usual practice to use unskimmed milk. It would be against the interests of the manufacturer to use impure or colored sugar, since these impurities in an article which forms the chief constituent of the food, and which can be bought in a pure condition at a low price, would produce a dark-colored, unsalable product.

And the same remark is true of the original milk itself. It is out of the question for the canner to employ a bacteriologist to examine the milk used, and the hardening and putrefactive changes which the condensed milk sometimes undergoes are usually due to failures in cleanliness, etc., at the dairy. Later on, the canner will participate with the public in the great benefits of a rigid system of inspection that will, first of all, begin with the dairy and the cattle themselves.

As things are at present, the manufacturer frequently has hundreds of cans which spoil in time on the shelves of the grocers from the causes mentioned. A small amount of contaminated milk contaminates all the rest used with it, and the results are not manifest until the goods are in the hands of the dealers. The canner is compelled to take them back very frequently, and often is most anxious to do so. Much of the residue is rejected by consumers as manifestly unfit for use, and so the dangerous and poisonous part is very small as compared with the total enormous output.

In this article I have avoided saying anything in relation to the effects on the health of children resulting from the use of milk containing more than half its weight in solid matter of cane-sugar—a foreign fermentescible substance. Nor in relation to the impossibility of preparing from such an article a modified milk, or humanized milk, which is comparable to woman's milk, or even feebly approaches it, in nutritive value.

These latter objections do not apply to the milk condensed without the addition of cane-sugar. But even more care should be used to see that the milk thus condensed is kept free from the products of bacterial action in the interval between milking and canning. And inasmuch as cane-sugar acts as a preservative, milk condensed without it is more susceptible to change and more apt to act upon the metal of the can than when cane-sugar is present. For these reasons it would be better were it preserved in glass bottles rather than in tin cans.

A CASE OF ANUS VULVALIS, WITH REMARKS ON CONGENITAL COMMUNICATION OF THE VULVA AND RECTUM.

By Thomas Dwight, M.D.,
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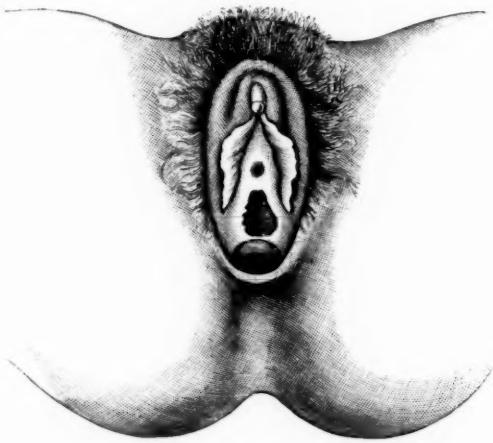
The following observation was made at the Harvard Medical School in March, 1894, on the body of a well-formed white woman, aged thirty-two years, apparently a virgin:

There was no anus, but the rectum opened into the vulva just above the posterior commissure by an orifice which would admit a large finger. The vulva was otherwise quite normal except that the hymen was poorly developed. It consisted of merely a slight fold above and at the sides; in fact it was quite indefinite. There certainly were no caruncule myrtiformes. No depression or suggestion of the place of the normal anus was detected before dissection, though carefully looked for, but after the parts had been removed from the body there was a distinct puckering of the skin about 2.5 cm. back of the vulva. There was no irritation of the skin about the vulva indicating incontinence of feces, nor was this observed during the three weeks the woman passed at the almshouse before her death.

It was, unfortunately, impossible to dissect the parts very thoroughly without destroying the specimen, but the following facts were observed. The rectum was smaller than usual. It ran from the tip of the coccyx along the floor of the pelvis, continuing tubular to the last, without a well-marked dilatation. The longitudinal muscular fibres seemed uncommonly developed. Although the woman was not fat, it might have required an incision three-quarters of an inch in depth to reach the gut from the puckered point. No sphincter could be detected at the termination of the rectum. The vagina was normal, and capacious above the entrance. The levator ani was very strong.

Cases of *anus vulvo-vaginalis*, as they are commonly called, are very rare, especially in the adult. Ahlfeld,[1] who uses this term, referring to Schröder's contention that the rectum never opens into the vagina proper, asserts that it does so, and that there may be a communication between the two, even when there is a normal anus. He goes on to say that the term *vaginalis* alone should be restricted to those cases in which the opening is evidently into the vagina and not in the region of the hymen and vulva. As the opening is usually nearly in the situation observed in this case, the term *vulvalis* seems the proper one. A tolerably thorough review of the literature allows the following statements: Almost all the cases reported are those of infants. Many of these



were successfully operated upon. The relatively small number observed in the adult implies that most of the infants either die or are successfully operated upon. Deutsh[2] reported the case of a woman of twenty-nine, with the rectum opening into the vulva below the hymen. There was a small pea-like appendage at the normal place for the anus, which felt like cartilage covered with skin. Dr. Henry Tuck[3] recorded the very interesting case of an American woman of twenty-six whom he attended in her confinement. A depression was felt at the normal site of the anus where the skin was deeply pigmented. The rectum opened about half an inch above the posterior commissure. It was closed by a sphincter, though rather a lax one.

[1] Die Missbildungen des Menschen.
[2] Neue Zeitschrift für Geburtshülfe, Bd. xxx. I have not seen the original.
[3] Boston Medical and Surgical Journal, 1876.

The patient had never had any difficulty in retaining the feces. In most of the children the normal position of the anus was to be recognized by a depression; but this was not constant. The size and shape of the opening into the vulva are various. It may be a mere pinhole and it may be valvular. Tuck is, I believe, almost the only one who speaks of a sphincter. The rectum seems usually to run along the floor of the pelvis, which is important in view of an operation for artificial anus. Provided the opening is sufficiently large for proper evacuations an operation is not imperative should the child's condition be unfavorable. This case as well as Tuck's shows that there need be no incontinence of feces. They give additional evidence that the feces accumulate in the sigmoid flexure. In a few cases there was more or less duplication of the internal organs.[1]

Most of the cases in which the rectum communicates with the vagina proper are those in which there is a double vagina and also a normal anus. The case to which Ahlfeld refers in his above-mentioned controversy with Schröder is that of Joseph,[2] who found in the body of a girl, three years old, in which both vulva and anus were normal, a double vagina, of which the smaller communicated with the rectum. The following remarkable case was observed by Caradec[3] on a living woman, aged thirty-two years. The anus and vulva were normal, except that the latter was rather shorter than usual, and the vagina was small. Between them was a third, slightly oval opening, 1.5 cm. before the anus, its longest diameter being antero-posterior. This led into a cul-de-sac lined with mucous membrane, which at its entrance offered a certain resistance, as of a sphincter. The anterior wall between it and the vagina was thicker above than below; the posterior wall, on the contrary, was thinner higher up and presented a fistulous opening large enough to admit a finger, which entered the rectum at least 5 cm. from the anus. The woman had suffered no inconvenience till after her marriage, when feces and flatus began to pass by the middle opening. Presumably this passage is to be called, as Caradec believes, a second vagina.

Finally there are excessively rare cases presenting a normal anus and an opening of the rectum into the vulva which is unsuspected during virginity. They are very important from a medico-legal point

[1] Since this paper was sent to THE JOURNAL an article by Dr. A. H. Buckmaster has appeared in the Transactions of the American Gynecological Society, vol. xix., 1894. In it he enumerates fifty-one cases besides that of his patient. Of these, fourteen were adults, including one aged sixteen. I was not acquainted with several of these cases; but, curiously enough, the list does not contain any of those I have mentioned. Though I do not speak by the book, I incline to think that the list could be considerably increased, perhaps nearly doubled. Of the fourteen adults it seems reasonably certain that six had control of the feces. The contrary is definitely stated only once. Probably the more happy condition is the rule.

[2] Beiträge zur Geburtshülfe und Gynäkologie, Bd. iii.

[3] Gazette des Hôpitaux, 1863.

of view. There is no doubt in my mind that the remarkable case which follows is of this nature. It was seen by E. Springfeld.[1] Shortly after marriage a woman of about thirty began to have feces pass by the vagina. Coitus was at first intensely painful and followed by moderate bleeding. The bridegroom had been drinking rather freely, but was not drunk. When the woman submitted to examination, which was a long time afterward, the place of the *fossa navicularis* was taken by an opening, a little more on the right than on the left, slightly patulous, admitting easily two fingers into the rectum. The borders were smooth, but an epithelial covering was not to be made out.

Before discussing the case it will be best to mention another, which, in this connection, is very instructive. Paul Reichel[2] operated on a multipara, aged twenty-five years, who since her marriage, three years before, had suffered from the escape of feces *per vaginam*.[3] The anus was normal, but the perineum was short, and the posterior part of the vulva abnormally deep. An opening large enough to admit a finger into the rectum was found just below the place for the hymen. The edges, except for some granulating fissures and scars, were covered with mucous membrane. There was no history of any injury or undue violence. Beyond question this opening was congenital. It was in the usual place for *anus vulvalis*, differing only from a typical case by the presence of a normal anus. The nature of the mucous membrane at the opening is conclusive. The preceding case, that reported by Springfeld, is of the same class. The position of the opening is the same, only its edges, which presumably suffered more violence, show somewhat less conclusively a continuous mucous membrane passing from the vulva into the rectum. It is not credible that this injury was done by the male organ to a normally built woman. Those who have learned the anatomy of the female perineum either on the living or by the study of frozen sections, know that the perineal body interposed between the rectum and the vulva makes such a perforation impossible. The only difficulty is to explain why no feces escaped the wrong way before marriage. It is possible that the opening was valvular, or guarded by a slight sphincter. It is also possible that there was no opening at all, but merely a thin septum, the imperfect correction of an early malformation which easily gave way. Springfeld seems to consider this explanation not unlikely. Be that as it may for his case, it does not apply to Reichel's. The true explanation in my opinion is the close apposition of the parts in a virgin, maintained partly by muscular tonicity, partly by the support of vascular structures.

[1] Vierteljahresschrift für Gerichtliche Medicin, etc., Bd. I., 1889.
[2] Zeitschrift für Geburtshülfe und Gynäkologie, Bd. xiv., 1888.
[3] As elsewhere, the word "vagina" is here used inaccurately.

REVIEWS.

CHIRURGIE OPERATOIRE DU SYSTÈME NERVEUX. Par A. CHIPAULT. Avec une Préface De M. Le Professeur Terrier. Tome Premier, Chirurgie Cranio-Cérébrale. Avec 431 figures dont 209 en couleurs. 8vo., pp. xx. and 744. Tome second, Chirurgie de la Moelle et des Nerfs. Avec 432 figures dont 365 en couleurs. Pp. 800. Paris: Reuff et Cie, 1894 and 1895.

OPERATIVE SURGERY OF THE NERVOUS SYSTEM. By A. CHIPAULT.

THE present work, when finished, will be the only complete work, we believe, on operative surgery of the entire nervous system, central and peripheral, in any language. The present part, the Surgery of the Head, Spine, and Nerves, consists of two volumes, that of the Surgery of the Spine (of which one volume has been published) of three or four volumes, the two parts supplementing each other very admirably. The author does not treat, of course, of those topics in general surgery which find a certain application to disorders of the nervous system, such as ligation of the carotid for cerebral hemorrhage, aneurism, or epilepsy, or treatment of nervous disorders by removal of the uterine appendages, etc.

The illustrations are very large in number (863), and, as a rule, admirable in quality, though many are rather rough outlines. One very striking point about them is that nearly all of them, even those which are adapted from other authors, are drawn by the author himself, showing that he is as skilful with his pencil as he is with his pen or his scalpel. The illustrations really illustrate. They show the development of the various instruments for cerebral surgery from the earliest days, including reproductions from some of the surgeries of the sixteenth century. One from Andrea a Cruce illustrates very curiously the condition of housekeeping at such a time. While the surgeon is in the midst of his operation of trephining the skull, and the ladies of the family are looking on, a cat in the corner has evidently just caught a rat, and is playing with it. Many of the illustrations are in colors, which make them more effective as well as attractive.

All of the important methods of cerebral localization are well illustrated and fully described in the text. As was observed in our former notice of the author's Surgery of the Spine (AMERICAN JOURNAL OF THE MEDICAL SCIENCES, March, 1894), the text of this volume also shows an exceedingly laborious and fruitful research in all languages and countries. There is no national pride which restricts his researches to the publications of his own countrymen, but he has drawn very freely from English, German, and American sources. In fact, no publication seems to be too obscure for him to have sought it out and recorded the results.

Under each important subject, such as intra-cranial tumors, osteo-

plastic resection, thrombosis of the lateral sinuses, laminectomy, resection of the fifth and of the facial nerves, etc., there are very complete tables of all cases of surgical intervention, with the sources from which they are derived. Besides this, at the end of each chapter is an extremely full and complete bibliography. These features will make this book necessarily a source from which subsequent authors will derive the greatest possible help in their investigations

At the end of the second volume there are full indexes of authors and an index of subjects which could have been greatly elaborated with advantage. Our French *confrères*, however, so seldom have *any* index, that we welcome this happy innovation, and hope others of his countrymen will follow his example.

Turning now to the text (to which space will allow only too brief a reference) the introduction deals with antisepsis, shock, and anæsthesia in the surgery of the nervous system. In these matters the author is fully in accord with the most advanced modern surgeons. He notes the observation of Dercum, that the frequency and intensity of shock become greater in proportion as we pass from the anterior brain toward the posterior, and thence toward the base and the cord. He states his disagreement with this general law, according to which interference with the spinal cord should be more dangerous than intervention in any other part of the nervous system. His belief, which is corroborated both by his figures and, we believe, by the general opinion of surgeons is, that this law is only true when restricted to the encephalon alone. Certainly, in our own experience, intervention in cases of spinal disease is much less dangerous than any equally serious intervention in diseases of the brain.

The author's statistics are also very striking. Analyzing 802 operations on the brain and cord, he finds that in operations on the anterior lobes (60 cases) the mortality is 1 to 60; in the convexity of the cortex (400 cases) 1 to 13; at the base (51 cases) 1 to 5; and in the cerebellum (37 cases) 1 to 3; whereas in the spine (117 cases) it is 1 to 17. It seems to us very natural that the shock should increase as we go from the anterior pole of the encephalon toward the base, because at the base and in the medulla oblongata lie those important centres for organic life, any lesion of which is excessively perilous—much more so than of those on the upper portions of the encephalon, which have to do with intellection, motion, and sensation. In 420 cases the percentage of mortality is identical in anæsthesia with ether, chloroform, or a combination of both.

In Chapter II., the Topography of the Brain, the methods of locating the fissures of Rolando and of Sylvius and the position of the middle meningeal artery are given very fully, together with such modifications as he thinks best.

The Wagner-Wolff method of osteoplastic trephining, together with the methods of filling a gap in the skull by celluloid or other foreign substances, and Koenig's method by chiselling the outer table away with the flap of scalp, are all noticed fully and illustrated, so that any surgeon can make himself familiar with the technique. Many cases also are quoted under this section and the next.

The only errors that we have noticed in the book occur here. The aluminum probe of Fluhrer on pages 273, 275, 285, and 292, and in the index appears as the probe of "Pflüger." On pages 302 and 303 a case, which is properly credited to Mills and Keen in the table on page 349,

is erroneously credited to Lloyd and Deaver; and on page 336, Case 24 is credited to Dercum and Heath, instead of Hearn.

The table of intra-cranial tumors which have been removed shows encouraging results. In 67 cases there were 47 recoveries, or 70 per cent. This shows that we have made distinct progress in the treatment of cerebral tumors, the mortality of former tables having been much larger than is shown by this more extensive table. *Per contra*, in exploratory operations in which either the tumor was not found, or being found was irremovable, there were 35 deaths in 47 cases, a mortality of nearly 75 per cent. This is due, doubtless, to the extensive interference with the brain which has often been practised in order to search for the tumor or to remove an inoperable one. Another important surgical lesson is that while the post mortem findings in cases of cerebral tumors would seem to show that perhaps not over 6 to 10 per cent. are operable, yet of 115 cases actually tabulated in 67 the tumor was found and removed. Of course, no case would be operated on which was not deemed possibly operable, which would explain to some extent the discrepancy between the surgical and the pathological findings.

The methods of operation on the ventricles of the brain are fully described, and the reviewer's paper of 1888 is credited with establishing the technique of the operation which has found a much wider and more beneficial application than was at first deemed probable.

The second volume consists of 128 pages on the Spine, and the remainder on the Surgery of the Nerves. The portion on the Spine, it would seem, might as well have been omitted in view of the elaborate treatment of the matter in the author's *Étude de Chirurgie Médullaire*, the complementary work to the present one. We must remember, however, that the present work practically is an Operative Surgery and does not pretend to take up questions of differential diagnosis and treatment other than the operative. No critical judgment, therefore, as a rule, is expressed in the book excepting from the operative point of view. Those desiring to learn the author's views on spina bifida and laminectomy, etc., must seek them in his work on Spinal Surgery proper.

The operative processes are very thoroughly described. The method of reaching all the nerves of the body is described and illustrated. Naturally, the surgery of the fifth nerve has a very prominent place; in fact, 324 pages (considerably more than one-third of the entire second volume) are given to the various procedures to reach its branches or the Gasserian ganglion. W. W. K.

A TEXT-BOOK OF PATHOLOGY, SYSTEMATIC AND PRACTICAL. By D. J. HAMILTON, M.B., F.R.C.S.E., F.R.S.E., Professor of Pathological Anatomy, Aberdeen. Two volumes, in 3 parts. 8vo., pp. xxii., 736; xxiv., 1139. London and New York: Macmillan & Co., 1889–1894.

PERHAPS there could be no apter commentary upon the wonderful progress of pathology during the past quarter century than a comparison of the large work before us with the small volume in which appeared Virchow's celebrated lectures upon cellular pathology, delivered during the spring of the year 1858. Then histology was in its inception. The cell had indeed long been known as the ultimate unit of living

matter, but its paramount importance as the active, controlling element of all the tissues was only beginning to be appreciated, and the greater part of the 440 rather coarsely printed pages of the first edition of Virchow's lectures is devoted to a demonstration of the part played by the cell in the normal tissues and of its importance in the production of the lesions of disease. Now, no such argument is needed. What Virchow presented as a well-supported theory has come to be universally recognized law, and we have passed on to a solution of the mysteries of the influences lying behind the cell and inciting it to activity. Thus, a work which shall present the present status of pathology must include a discussion of the known facts regarding the causation of disease, it must relate all the discoveries of hundreds of workers in the vast field of pathological anatomy and histology, and it must contain for the clinician a rational explanation of the symptoms which he is daily studying at the bedside, so that he may be enabled to read through them back to the condition of which they are the outward manifestation. It is, then, no holiday task, the compiling of such a work, and it is not to be wondered at that about five years should have elapsed between the publication of the first and second volumes of Hamilton's *Text-book*.

As the work is not intended simply as a treatise on pathology, but as a working manual also, it opens with a detailed description of the autopsy, of the most approved instruments for its performance, and of the manner of its conduction. The various methods of preparing the tissues for microscopic examination are then detailed, the microscope is described, together with the uses of the various accessories, drawing and measuring apparatus, immersion lenses, etc. The way is thus prepared for the consideration of the causation of disease in general, and we are introduced to the methods of practical bacteriology as affording the means of investigating the problems concerned in it. The culture-media and the methods of their preparation, the special apparatus of the bacteriological laboratory, the making of cultures, the separation of species, the staining and study of the bacteria, animal inoculations, immunity, disinfection, etc., are all treated in detail.

Part II. is devoted to the general pathological processes, as they are called, and includes the discussion of the various infiltrations and degenerations of tissue, of inflammation, suppuration, healing and organization of tissues, ulceration, and dropsy.

Part III., devoted to the diseases of the various tissues and organs, makes up the bulk of the work, beginning at page 347 of Vol. I., and running through 953 pages of Vol. II. A description of the structure and reproduction of cells in general introduces the discussion of the various "new formations" and tumors, included among which are the so-called infectious granulomata—*i. e.*, tubercle, lupus, and gumma. One hundred and seven pages are allotted to the blood, after which the pathology of the various organs is discussed, beginning with the heart and bloodvessels and ending with the diseases of the skin. In this part, also, are chapters on the various malformations and on the diseases of the fœtal membranes and placenta.

Part IV. treats of the diseases due to vegetable and animal parasites, and in it the bacteria are studied systematically in their relation to the various diseases of which they are the cause. In this section are found also many of the discoveries of the past five years in general bacteriology, facts which would properly have been discussed in Part II. had they

been known at the time of the publication of Vol. I. This is important, since the most material advances in pathology during the past few years have undoubtedly been in this very field, and by thus including them at the end of his second volume Hamilton has brought his book well down to date, and has in a very great degree bridged the gap between the publication of the first and second volumes.

The closing pages of the work (Part V.) are devoted to a discussion of animal heat and fever.

When it is added to this synopsis that the work is well and abundantly illustrated (623 figures, including a number of colored plates), that the numerous references in the text are supplemented by bibliographical lists at the end of every section, and that throughout the work the relationship of pathological anatomy and pathological physiology, of lesion and symptom, is sought to be elucidated to the fullest extent, it will not be doubted that we have here the most ambitious work upon pathology which has issued from the English press. A particularly commendable feature of the book is the author's effort to reduce all his explanations of pathological processes to conformity with physical and chemical law. We are too apt to overlook the fact that the laws of these sciences, which we recognize as governing all physical and chemical phenomena outside the body, apply also to similar processes within it, and oftentimes afford explanations of otherwise obscure conditions. The truth of this is apparent after the perusal of Hamilton's chapters on inflammation, exudation, œdema, and on the disorders of respiration, circulation, and nutrition. We must also mention the brief summary of the anatomical and physiological characteristics of each organ which precedes the discussion of its lesions, particular stress being laid in each instance upon those structures and functions most disordered by disease.

It would be surprising, indeed, if in so large a work there were nothing found to criticise. Pathology is now so extensive a subject that it is almost inconceivable that perfect unanimity of opinion upon all points should exist among its votaries, and it is with no desire to detract from the general commendation which Hamilton's work deserves that we call attention to two or three particulars in which we cannot entirely agree with him.

First and foremost, we must take exception to the classification of tubercle and gumma among the tumors. Even our German *confrères*, who have always inclined to regard the neoplastic elements of these processes as their most important characteristics, stop short of this, making for them a special class to which they apply the name "infectious granulomata." But, even this appears to us illogical and inducive of great misconception of the real nature of the process underlying these conditions. Nothing could be more clear than their inflammatory nature, and we appreciate no reason for their dissociation from other inflammatory conditions induced by bacteria which it is the universal custom to discuss under the general heading of inflammation. Tubercle and gumma surely only differ from these in the degree of participation of the various structural elements which are always involved to a greater or less degree in all inflammations. Is not the wall of granulation and cicatricial tissue which forms about every retained necrotic or suppurative focus, separating it from the surrounding healthy tissue, as much a new growth as the connective tissue developed in connection with tuberculosis? And may we not as consistently consider the lesion

of tuberculosis to have its distinctive structural characters because of distinctive peculiarities in the virus by which it is induced, as to suppose the excessive emigration so characteristic of suppurative inflammation to be due to the pronounced chemotactic quality of the micro-organisms of suppuration? The writer has, in fact, found it easy for students to conceive the idea of qualitative differences in the various inflammatory processes of bacterial origin being dependent upon qualitative differences in the various bacterial toxines where great difficulties have attended other conceptions of their production. In every inflammation the elements of the tissue participate, in a few degenerating only at the outset, in most showing sooner or later some tendency to proliferate. Now, when we remember that recent investigations into the nature of the tubercular virus have shown one of its ingredients to possess a remarkable power of stimulating connective-tissue cells to growth, and that in the slow course of the majority of tubercular processes abundant time is afforded for such connective-tissue development as occurs, those elements of the tubercular process which are usually cited as making it essentially different from other inflammations are explained. It would seem, therefore, to the writer that the separation of tubercular and syphilitic processes in a class by themselves is a distinct hindrance to the simplification of our conceptions of inflammation made possible by the developments of recent years in our knowledge of their etiology.

Again, we would take exception to the attribution of the power of spore-formation to the tubercle bacillus. It is true that many allusions to spore-formation by these bacteria are scattered through the voluminous literature upon them, but so far as we know no single element of proof of its occurrence has ever been produced, the only basis for such an assumption being a beaded appearance, often observed in the bacilli of tubercular sputum. But this may be much better explained upon the theory that such bacilli are dead and undergoing disintegration, both because of the irregularity of the beading and because of the presence of several lighter spots, the so-called spores, in each bacillus, while, so far as is known, in the definitely spore-forming bacteria only one spore develops in a single germ.

We must also express surprise that Hamilton should have selected Friedländer's pneumococcus as the cause of lobar pneumonia in preference to the Fränkel-Weichselbaum germ so constantly met with in that disease and now so universally acknowledged as by far its most frequent cause. And, finally, what is an "oat-seed-like spindle-cell sarcoma" (Cf. vol. i. p. 365)? Its source is not given, and, though we are loath to suggest it, the appearance of the cells as depicted in the figure is decidedly like that of some leiomyomata which we have seen, most of whose cells had been cut obliquely in the preparation of the section.

But, as has been said, the book is in general to be much commended, and affords many valuable suggestions to the teacher of pathology, while being at the same time a useful work of reference for the student and for the clinician. Its typography and general make-up have all the excellences of Macmillan's publications. J. S. E.

A Clinical Manual of Diseases of the Eye, Including a Sketch of its Anatomy. By D. B. St. John Roosa, M.D., LL.D. With one hundred and seventy-eight engravings and two chromo-lithographic plates. New York: William Wood & Co., 1894.

The task of the fault-finder is not an enviable one; yet there are occasions when to find fault becomes a duty which one cannot conscientiously put aside. Having undertaken to review Dr. Roosa's recently issued *Manual of Diseases of the Eye*, we find ourselves, very unexpectedly, in the presence of such an occasion. To say of the book, without qualification, as a critic once said of phrenology, that what is new in it is not true, and what is true is not new, would be unwarrantable; yet we are forced to admit that an impression closely akin to this was uppermost in our mind when we closed the volume after a somewhat careful examination of its contents. In a word, where the author has followed the beaten track he has told us what we already knew, and told it in a less satisfactory manner than it has been told before; where, on the other hand, he has ventured upon bypaths of his own hewing—where he has advanced views that are peculiarly his own, he has proved himself to be an unsafe guide—the views proclaimed are, more often than not, radically unsound. Furthermore, the book gives evidence, from beginning to end, of want of accuracy and care upon the part of its author. This is shown in the unsystematic arrangement of the volume, in looseness of statement, in incorrectness in quotation, in carelessness in literary construction, and in the seeming absence of proof-reading and revision. These are sweeping statements, which should not be made without abundant justification. Let us see if such justification exists.

On pages 263 and 275 we find these statements, which seem to us about equivalent to saying that most cases of smallpox have their origin in neglected eczema: "Much trachoma has occurred from neglected catarrhal conjunctivitis." "Trachoma is generally a chronic disease, the result of a conjunctivitis that has run its course. Investigation usually shows that there was at first an ordinary catarrhal conjunctivitis from which this sprang." Compare with this the clear-cut statement of Fuchs, in the recently issued English translation of his *Text-book of Ophthalmology* (p. 66): "Trachoma originates exclusively in infection proceeding from another eye affected with trachoma. Infection takes place by transfer of the secretion; contagion by means of the atmosphere, the existence of which was formerly accepted, seems not to occur."

On page 317, under the heading "Sympathetic Irritation of the Fellow-eye," we read: "This affection should be carefully distinguished from sympathetic *inflammation*;" but, on the following page, this statement occurs: "It is generally believed that it is the contraction of a cicatrix, or a new inflammatory process about a foreign body, which excites the sympathetic irritation. If we accept in full the microbic origin or septic origin of the disease, it is due solely to the transference of septic material from one eye to the other." The caution given on the preceding page seems to have been lost sight of; for, we believe, even the most ardent advocate of the theory that sympathetic *inflammation* is

due to the migration of septic organisms from the exciting to the sympathizing eye has never claimed that sympathetic *irritation* originates in any such fashion.

On page 412 we find a repetition of this confounding of the two conditions by the author. In speaking of the different forms of incision which have been employed in cataract extraction, he describes that of Von Gräfe as encroaching too much upon the ciliary region. "This, he says, "was to invite sympathetic irritation, and so it proved. More eyes were lost from sympathetic irritation during the days when this operation was being performed than at any time before or since." And again on page 421 we read: "The wound made in the sclera by the bident [Dr. Agnew's] is a real objection to its use, because such an incision may possibly cause sympathetic irritation."

In the description of catarrhal conjunctivitis, on page 260, we find this: "It is a disease which sometimes is epidemic in communities. It is generally known as 'pink-eye' when this occurs. Spring catarrh is also a name used by the Germans to designate this epidemic form of catarrhal conjunctivitis." It is hardly necessary to say that the "spring catarrh" of German writers (which all must admit *is* an ill-selected name) bears about as much resemblance to catarrhal conjunctivitis as pterygium does to keratitis punctata.

On page 373 the unqualified statement is made: "Cataract is really an inflammation of the lens (phakitis)." And then follows, as though it were conclusive evidence in support of this assertion, this: "When mechanical injury has produced cataract, or, rather, is producing the disease, we may distinctly note the swelling that characterizes a true inflammatory process." Fuchs says, and we believe this view is entirely in accord with the teaching of modern pathology and with our present conception of what constitutes inflammation, that "inflammation of the lens—phakitis—does not exist. Inflammatory elements, such as round cells, occurring in the lens, do not originate in it, but enter the lens from the outside through an opening in the capsule."[1]

On page 535 we are told "presbyopia is a condition dependent upon the accommodative power, a *disease* [Italics our own] of the accommodation." On the following page, however, we find that, after all, the author probably does not regard presbyopia as a *disease*, for he says, "The *physiological* [Italics again our own] causes of presbyopia, in the light of our present knowledge, may be said to be of a threefold character." And then he proceeds to discuss the three causes. The first, to which he seems to attach much more importance than to the two others, and to which he devotes more than a page (seven lines being sufficient for the consideration of the second cause—loss of elasticity of the lens), is described as "a diminution in the contractile power of the ciliary muscle." "With the dawn of youth," he tells us, "its [the ciliary muscle's] power begins to decline, especially if over-exerted and if continuously employed. . . . . The aboriginal inhabitants of our country, the frontiersman, the scout, the sailor, the country gentleman leading an out-door life, usually have a greater accommodative power . . . . than the student . . . . the bookkeeper and the man of letters. To this there may seem to be exceptions, but usually they are only apparent ones, or they really prove the rule, that excessive use of the

[1] Op. cit., p. 368.

accommodation for near objects brings on presbyopia at an early age." To most of us, we venture to predict, all this is "news," indeed. The commonly accepted view of the nature of presbyopia is so well put by Fuchs, that we are constrained to quote him once more. "The diminution in the accommodation," he tells us (*op. cit.*, p. 622), "cannot be referred to the decrease in the power of the muscles in general, and the ciliary muscle in particular, occurring in old age, for it begins in youth, and probably even in childhood—that is, at the time when the muscles are still gaining in strength. In fact, the cause of the diminution of the accommodation lies in the gradual decrease of the elasticity of the lens."

The third cause of presbyopia Dr. Roosa describes as follows: "There is, perhaps, also an absolute flattening of the lens, on account of increased density (Stellwag) which shortens the axial diameter of the eye, [*sic*] and constitutes what Donders called acquired hypermetropia."

The beginner in ophthalmic practice, having been told on page 15 that "the nasal duct in its course passes downward, backward and outward," and that "this fact should be kept in mind when passing the lachrymal probe," will, we take it, be somewhat disconcerted when he reads upon page 206 that the probe should be "passed downward through the sac, and then downward, outward, and forward through the duct into the nose."

In this connection we would express our surprise at the author's statement, on page 248, that he occasionally finds it necessary to employ a "Number 20" lachrymal probe; for we have a distinct recollection that at the International Congress of Ophthalmology which met in Edinburgh last summer, when the subject of the employment of large lachrhymal probes was under discussion, he was most emphatic in condemning their use.

In the chapter devoted to "inflammatory affections of the conjunctiva" the author describes, besides catarrhal conjunctivitis and purulent conjunctivitis, an affection which he calls "blennorrhœa of the conjunctiva" (p. 262). We are unacquainted with such a disease as he portrays under this heading, and it seems evident that in describing it he simply had in mind severe cases of catarrhal, and mild cases of purulent, conjunctivitis. The name (blennorrhœa), it may be remarked, is a distinct misnomer when employed as a synonym for purulent ophthalmia (as the Germans use it), for its literal meaning is a flow of *mucus*.

Under the title "myotics," eserine, physostigmine, and pilocarpine are mentioned, and are said to be "used as local applications in solutions of one to four grains to the ounce" (p. 185). As to the first named, the statement would have been much nearer correct had it read from *one-eighth grain* to four grains to the ounce. On the same page, under the antiquated title "irritants," calomel, red and yellow oxides of mercury, and jequirity are described. The red and yellow oxides of mercury are said to be applied to the eye in the form of ointments, varying in strength from half a grain to two grains to the *ounce*. The red oxide, we believe, is but little used at the present time; the yellow oxide, for application to the eye, is commonly used in the strength of one grain to the *drachm*.

In the tabulation of the symptoms of acute glaucoma (pp. 355 and 356) no mention is made of the shallowing of the anterior chamber, of the enlargement of the pupil, or of the greenish pupillary reflex from which the disease derives its name.

The opinion expressed on page 384, and reiterated on page 400, that

when a cataract is allowed to become over-ripe it is apt to induce choroiditis, atrophy of the iris, softening of the globe, etc., is one which, we believe, has the merit, at least, of novelty. The advice given on page 223, that, to induce anæsthesia in operations for cataract, an eight-grain solution of cocaine should be dropped freely on the cornea "every three to five minutes," for "from fifteen minutes to a half-hour before the operation," we regard as decidedly unsound. There is a consensus of opinion upon the part of experienced operators that such prolonged use of cocaine is not only unnecessary, but is actually harmful and should be avoided.

"The labors of renowned men," says the author in his preface, "have settled certain points forever; but there are still departments of ophthalmic science and art in which many things are under discussion, and concerning which widely differing views are held, especially in the United States. For my views on these subjects, which are chiefly to be found in the fourth part of this work, I invite an impartial hearing." The fourth part of the work, to which our attention is thus especially directed, is that in which errors of refraction and accommodation and affections of the ocular muscles are treated of. Here it is that the author, with a hardihood worthy of a better cause, ventures most frequently and most radically from the beaten track and indulges in bypath-hewing with the unhappy results alluded to at the beginning of our review.

Asthenopia he divides into "true or local, and the false or symptomatic." True asthenopia is that "which depends upon a refractive anomaly." The symptomatic or false asthenopia "depends on some error in the general nutrition, neurotic constitution, nervous exhaustion, or the like" (p. 448). *Muscular* asthenopia *has no existence.* He "formerly accepted the ordinary classification of an asthenopia dependent upon insufficiency of the external ocular muscles," but he has "come finally to reject it altogether" (p. 450). Even the term, he thinks, should be abolished, "as having no rightful place as generally used in ophthalmology" (p. 520). He no longer measures the relative power of the ocular muscles, and declares that we have "no occasion to look to insufficiencies as faults needing correction, except when they cause deformity or destroy binocular vision" (pp. 451, 452). "When I became familiar with the use of the ophthalmometer, in 1888," he tells us on page 520, "I once for all abandoned the idea of relieving muscular asthenopia except by correction of an error of refraction," and, he adds, "if the student will carefully correct the errors of refraction he will have no need for tenotomies except for strabismus, nor for prisms except for paresis or paralysis of the external muscles."

The only comment we will make upon such remarkable statements as these is to say that between one who holds such views and permits them to influence his practice, and one who cuts the ocular muscles for lateral insufficiencies without knowing anything of the refractive condition of the eyes, there is but little to choose. One is as much an extremist, is as distinctly eccentric, as the other.

Although we realize that our notice of Dr. Roosa's book has already reached an unreasonable length, there are a few other points in the part of the work under consideration which we think should not be passed over in silence. One of these is the statement made on page 453, that the best thing to do for the "large number of American asthenopes . . . who usually have from one to one and one-half diopters of

corneal astigmatism, with from one to three or four diopters of hypermetropia, . . . is to make rather light of their troubles, and correct the astigmatism." If *three or four diopters* of hypermetropia, in an asthenopic individual, are to be "made light of," and disregarded in the adjustment of glasses, we should like to know at what point, in the opinion of the author, hypermetropia really becomes of consequence—not much short of aphakia, we should suppose. Of a piece with this is the opinion expressed on page 468, that the prescribing of convex glasses, for distant vision, in facultative hypermetropia is bad practice, and that it results in the relief of asthenopia only "in entirely exceptional cases."

We are not so much surprised, however, at these shortcomings upon the author's part in dealing with the correction of hypermetropic errors of refraction when we find, upon turning to page 465, that he actually does not know what the meaning of the term manifest hypermetropia is. "Manifest hypermetropia," he there tells us, "is that form in which the vision for the distance is improved by convex glasses." It is hardly necessary to say that this definition of manifest hypermetropia is absolutely incorrect. Distant vision of normal acuteness, without the aid of convex glasses, is not infrequently present, even in high degrees of manifest hypermetropia; indeed, in early life, this is the rule rather than the exception. We had marked for criticism a number of other passages in the chapters upon refractive and muscular anomalies, but want of space compels us to omit their consideration.

We have found fault with the literary style of the work; one or two examples must suffice to justify this criticism. On page 154 we are told: "The ophthalmometer, as invented by Helmholtz, was used by Donders and Knapp, in the first investigations of astigmatism since the days of Dr. Thomas Young." As Dr. Young died in 1829, when Donders was not quite eleven years of age, and Dr. Knapp was not yet born, this statement, it must be admitted, is a little mystifying. But hardly more so than this, on page 408: "It is true, that among Arabs and Persians and in the seventeenth and eighteenth centuries, cataracts were extracted by German and French surgeons." Or this, on page 421: "In one case Dr. Webster was obliged to remove the bident before he could remove the lens, although in some cases enucleation must be resorted to, in a dislocation of the lens, and in others the removal of the lens is only followed by severe inflammation which necessitates subsequent enucleation." The expression, "in quite a proportion of cases," occurs on page 128, and on page 307 we read: "The immobility of the iris is sometimes masked, so that we are not aware that it has suffered until a mydriatic is used, and we find it very sluggish." Of a different character are these (p. 546): "Although all deviations of the eye . . . *is* properly called strabismus;" (p. 535) "In advanced age, not only the near point but also the far point *recede*;" (p. 502) "It will be seen that not one of these who had no hypermetropia by the ophthalmoscope *were* without corneal astigmatism."

Two important quotations are made from Donders's *Accommodation and Refraction of the Eye*, one on page 508, the other on page 455. An error in punctuation in the former renders the quotation meaningless, while the latter not only contains a similar error, which destroys the sense of the last part of the quotation, but also a grammatical one ("ensues" for "ensue") which neither Donders nor his translator is responsible for.

Typographical errors present themselves with inexcusable frequency.

Among others, we have noted the following: on the title-page a "g" takes the place of the final "c" in "lithographic." In the index (p. x.), page "191" should be "119." In a quotation from Loring (p. 86), two errors occur, "bow" for "bowl" and "vein" for "rim." On page 247, "then the probe is *taken*," evidently should read "taken out." "In" takes the place of "is" on page 308; "correction" of "condition" (p. 404); "pen" of "pea" (p. 421); "preached" that of "reached" (p. 464). Other errors are "amblyopia" for "myopia" (p. 473); "occulist" for "oculist" (p. 523); "insufficiencx" for "insufficiency" (p. 525); "causes" for "cases" (p. 526); "unimpaired" for "unimproved" (p. 539); "or" for "for" (p. 541), etc.

Having indulged in so much unfavorable criticism of Dr. Roosa's book, we are glad to be able to say, before bringing our notice to a close, that there are some things in it which we can heartily indorse. Among these we may mention his condemnation of optico-ciliary neurotomy (p. 222); the advice given on page 247 to pass a probe through the canaliculus as a preparatory step to slitting it, in the treatment of stricture of the nasal duct; the opinion expressed on page 402, that local causes, such as the accommodative strain induced by astigmatism, are, not infrequently, important factors in the causation of cataract; and his commendation of the administration of sulphonal after cataract extractions, for the purpose of quieting the nervousness of the patient and insuring a better night's rest (p. 405). On page 549 there is reported a case of amblyopia developing in a squinting eye, which eye was known to have had normal vision previous to the occurrence of the squint. The case, which happened in the author's practice, is related because of its important bearing upon the question of the origin of the amblyopia of squinting eyes, and it is certainly one of great significance.

S. T.

MEDICAL JURISPRUDENCE, FORENSIC MEDICINE, AND TOXICOLOGY. By R. A. WITTHAUS, A.M., M.D., and TRACY C. BECKER, A.B., LL.B. Vol. I. New York: William Wood & Co, 1894.

IT must be confessed, in view of the admirable works of Devergie, Tardieu, Casper, Taylor, Christison, Woodman and Tidy, Wharton and Stillé, etc., accessible to every student, that the *raison d'être* for an elaborate work on medical jurisprudence, forensic medicine, and toxicology does not seem obvious. Apart from this consideration, it is particularly difficult, however kindly disposed the critic may be, to do justice to the merits of an extensive work such as the above, of which but one volume has appeared. The main objection to the present volume, as is usually the case in all such "systems" in any department of medicine, is that too much attention is given to relatively unimportant subjects and too little to important ones. Thus, for example, while 46 pages are devoted to the consideration of "The law of evidence concerning confidential communications," and 158 pages to the "Synopsis of the laws governing the practice of medicine" in the United States, Great Britain, etc., only 105 pages are given to death from suffocation, hanging, strangulation, and drowning, subjects which appear to us at least far more worthy of consideration than the laws governing confidential

communications and the practice of medicine in Manitoba, Prince Edward's Island, and Ireland. On the other hand, the "powers and duties of coroners" have been so often discussed by practitioners that we welcome the excellent and authoritative article defining the functions of that ancient official. To those, however, who ever have held the position of coroner's physician it will certainly strike as extraordinary that in the article "Medico-legal autopsies" the subject of the identification of the deceased by two witnesses who personally knew the deceased during life is never mentioned.

This illustration, like many that might be given, gives the impression that the different articles in this work have been prepared by very learned jurists and physicians and surgeons of the highest standing, but not by men who have passed their lives in morgues and courts of justice.

This theoretical tendency is seen on the very first page of the introduction to the work, where the wordy and superfluous distinction between medical jurisprudence and forensic medicine is made, the former being defined as "medical law," the latter as the "application of medical, surgical, and obstetrical knowledge to the purposes of legal trials," Zacchias being referred to as the "father of medico-legal science," and as having recognized this distinction without a difference. Apart from Zacchias not being the first to write on medico-legal subjects, the fact that in the work *Pauli Zacchiæ Romani Quæstionum Medico-Legalium Francofurti ad Moenum*, MDCLXXXVIII, a page of advice is given to the *lectori medico* and another, with becoming modesty, to the *lectori legum perito* does not warrant the statement that Zacchias recognized any such difference as that made above. A lawyer may look at a trial from the legal standpoint, and the doctor from the medical one—the accused is, however, only tried once. In the article on "Identification of human bones," the statement that the skull of a fossil batrachian compressed into the rude resemblance to the human cranium will excite comment by those who are familiar with the writings of Scheuchzer and Cuvier. It is well known that the famous Oenigen fossil, described as "Homo deluvii testis et theoscopos" consisted of the remains of the skeleton of a batrachian, not simply of the head. In the otherwise excellent article on the "Medico legal determination of the time of death," it is to be regretted that in considering the subject of the loss of heat no mention is made of the classical observations of Goodhart, Niderkorn, Burman, etc., and in describing the phenomena of rigor mortis the work of Sommer should not be mentioned. It is true that the work of Sommer, *De Signis Mortem Hominis*, etc.: Hauniae, 1833, is a very rare one; nevertheless a copy of that book is to be found in Washington. In the article on "Wounds" we are surprised to see any positive statement as to the character of wounds made by "china, or glass, or fragments of bottles," as it is often impossible to say whether a wound was made by such fragments or by a cutting instrument. The article on "Gunshot-wounds" is an excellent one, as might be expected, emanating from such a high authority. That on the "Medical relations of electricity" is a most valuable and important contribution to the literature of that subject, the execution of criminals by electricity having given physicians the rare opportunity of observing the phenomena presented in death from that cause. The work generally, the contents of which we have endeavored to give some idea, is well printed, and many of the illustrations are most excellent. We regret to say, in conclusion, that however interesting this

work may prove to the general reader, for the special student of medical jurisprudence it is not sufficiently full, the literature of the subject being but rarely referred to, while to the expert, medical or legal, actively engaged in the practice of their professions the work, even if never completed, is too voluminous. H. C. C.

DOSE-BOOK AND MANUAL OF PRESCRIPTION-WRITING. By E. Q. THORNTON, M.D., Ph.G., Demonstrator of Therapeutics in the Jefferson Medical College of Philadelphia; Acting Assistant Surgeon in the United States Marine Hospital Service. Pp. vii. 334. Philadelphia: W. B. Saunders, 1895.

THE book is an excellent presentation of a subject which is usually imperfectly taught in the schools if we may take as evidence the prescriptions found in the journals and reports of society proceedings. In the first part the weights and measures, both English and metric, and the methods of conversion from one to the other system are clearly set forth and well within the comprehension of the average medical student. Prescription-writing proper occupies the second part. We heartily indorse the arguments for the use of Latin, and believe that the grammar, vocabulary, and general directions will be found to be ample for the acquisition of correct phraseology. The subject of incompatibility is illustrated by excellent examples; antipyrin might have been added to the list of substances mentioned.

Finally, the table of solubilities will be of frequent use, especially to the younger practitioner. The third part takes up the official preparations and methods of prescribing. The preparations are classified as aceta, tincturæ, vina, etc., the doses given in both apothecary's and the metric systems, and illustrative prescriptions appended. The question of dosage and methods of administration receives attention in the fourth part, which closes with an extended list of official and officinal drugs and preparations, alphabetically arranged, and under each heading the substance is defined, its dose given, its preparations named, and occasionally remarks on its preparations or use.

In the appendix are to be found questions intended to test the knowledge of the student, and their answers. We have carefully examined this book and have found it to be singularly accurate, complete, and well adapted to the use of the student and junior practitioner. Of especial value we would note the insistence and example of the author in the avoidance of all abbreviations and the substantiation of his claim that this book is based upon the Pharmacopœia of 1890. R. W. W.

PROGRESS

OF

MEDICAL SCIENCE.

THERAPEUTICS

UNDER THE CHARGE OF

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Antitoxin and Nephritis.

Dr. Lennox Browne reports that a further experience in the use of this remedy has resulted in six deaths out of a total of eight cases treated, all due to inflammation of the kidneys. This is in confirmation of the testimony of Benda, who found, on the necropsy of thirty-nine subjects dying under the antitoxin treatment, nephritis in thirty-three cases; of these, eight were of a severe grade. From an analysis of one thousand consecutive cases of diphtheria, death, under the usual treatment, resulted from uræmia in only two and seven-tenths per cent., the total mortality being at the rate of twenty-seven and four-tenths per cent. It is clear that if we are to get at the truth of the results of the antitoxin treatment a necropsy should be made on every case dying under it, that we may learn the exact cause of death in each instance of fatal issue.—*Medical Press*, 1895, No. 2905, p. 51.

Diphtheria; Relapses after Treatment by Behring's Antitoxin.

Dr. Carl Goebel calls attention to the three hitherto-reported cases; one, that of Kossel, which died on the day of the relapse, four weeks after the first attack, of gangrenous diphtheria; the amount of serum which was used is not stated. The remaining cases received, each, one hundred and sixty immunizing units. To these the writer adds two cases. The first received fifteen hundred immunizing units. A boy, twenty-five months old, was attacked on October 22d, the membrane being found on both tonsils. Tracheotomy became necessary one-half hour after the injection, and the canula was retained until November 5th, when convalescence was established. On December 1st the diphtheric membrane again appeared on tonsils and hard and soft palate. On the 18th the condition was as before the relapse.

Evidently in this case a relapse occurred so late as forty days after the use of the antitoxin. In the second case, a girl of seven years became ill on August 30th. Recovery was established four days later. On September 30th a tonsillar diphtheria was found, which ended on October 5th. In this case a relapse occurred twenty-eight days after the primary attack, which was treated by the injection of one thousand immunizing units.—*Deutsche medicinische Wochenschrift*, 1895, No. 2, S. 29.

Nuclein.

Dr. Frank W. Garber states that we have reason to believe that this substance possesses the power of stimulating the production of white blood-corpuscles, which are the natural defenders of the body, and that aside from this power they are also valuable as germicides. If the red blood-corpuscle is derived from the white, then nuclein will stimulate the growth of blood-corpuscles, and will be an almost direct enricher of the blood-supply. Clinical experience has certainly shown that nutrition is improved under the use of this treatment. It promises to be useful in nervous disorders which are not of organic origin, and in troublesome cases of digestive disturbances. It is possible that a large share of the good results alleged to have been obtained from the use of the so-called animal extracts may be properly attributed to the possible nuclein which they may contain. In the cases reported the drug was given once daily hypodermically, the dose varying from fifteen to forty-five minims; for the most part thirty minims were the maximum dose. The injections were made into the muscular tissues as near the site of the tuberculous lesion as could be, although this does not seem to be essential; they were often given in the arms; ordinary antiseptic precautions were observed. The needle should be frequently sharpened. Rubbing the part seems to cause a more speedy absorption of the solution and to lessen the period of pain, although the latter is never lengthy nor severe. The part, from frequent injections, becomes larger and more insensible; abscess has never resulted. Twelve cases were reported, the majority being of pulmonary tuberculosis, and these reports seem to warrant the belief that it is an agent of value in incipient tuberculosis, but that little can be expected of it in advanced cases. We are not in a position to say that it is positively curative. —*Therapeutic Gazette*, 1895, No. 1, p. 5.

The Treatment of Acute Coryza.

Dr. Marcel Lermoyez proposes for the aborting of this disease, in the early stages, the inhalation of ten drops of the solution of pure carbolic acid, 5; ammonia, 5; alcohol, 10, and distilled water. Of the snuffs, that of menthol, 1; cocaine hydrochlorate, 2; salol, 20, and boric acid, 60, finely powdered, has the preference. Of the internal remedies, a mixture of equal parts of tincture of belladonna and tincture of fresh aconite root, of which thirty drops can be taken in two doses. A steam bath to produce energetic sweating sometimes has an immediate effect. Among the palliative measures may be mentioned spraying with warm solution of cocaine hydrochlorate, 1 per cent., to keep the nostril open. Or an unirritating antiseptic powder may be used, as cocaine hydrochlorate, 2; menthol, 1; bismuth salicylate,

20; milk-sugar, 20. If there is reason to fear cocaine-poisoning, a 5 per cent. solution of menthol in olive oil, sprayed into the nose, may be used. For relieving the facial neuralgias, and for the chilly sensations and malaise, at each meal may be taken quinine hydrochlorate, four grains, with antipyrine, eight grains. For the erythema of the nasal orifice, which is caused by the discharge, borated vaseline is useful. Hygienic measures, as sojourn in his room, and the avoiding of violent blowing of the nose, or sneezing, which may force septic mucus into the middle ear, should be observed. Douching the nose is absolutely forbidden during the acute stage, because it irritates the mucous membrane and endangers the ear. If later there is a chronic muco-purulent discharge, douches are indicated. Prophylactic measures consist in guarding against imprudence and in curing the chronic lesions of the nose.—*Journal des Praticiens*, 1895, No. 4, p. 51.

Injections of Artificial Serum.

Dr. Gingeot makes use of 5 per cent. of sodium phosphate and 10 per cent. of sodium sulphate, in solution in distilled water, in neuropathic cases. He reports a single successfully treated case in which the diagnosis was onomatomania, agoraphobia, *folie du doute*, arithmomania, attacks with loss of consciousness, hysterical hæmianæsthesia, and cranial malformation. The results were not due to suggestion, since various other methods had completely failed. The amount used is from fifteen to fifty-two minims.—*Journal des Praticiens*, 1895, No. 2, p. 17.

The Treatment of Pulmonary Tuberculosis.

Dr. Karl von Ruck recommends exercise, during the hours when the patient is free from fever, without reaching fatigue or shortness of breath. When the temperature rises above 100° F. he should go to bed in a well-ventilated room, and remain until it has subsided. On the following day he should anticipate the rise by going to bed one hour earlier. If the temperature is 101° F., an ice-bag should be applied over the heart until it is reduced to below 100° F. When there is pulmonary congestion, irritable cough, or pain in the chest, a cold, wet compress encircling the chest, covered with dry flannel, should be applied at bedtime and remain all night. A cold sponge-bath, followed by vigorous dry friction, must be given every morning, menstruation being no contra-indication. In the absence of fever his diet should be mixed and generous, and if he has not sufficient appetite, milk must be given freely between meals. Fever patients, as a rule, should receive only liquid food, with bread and butter added, while the temperature is elevated; the heartier meals must be given when fever is absent. If the weather is favorable, he should be out of doors; not, however, constantly upon his feet, or in a carriage, or on horseback, conditions which come within the limit of exercise, but quietly sitting or reclining upon a porch or place protected from the wind and dust, the latter being always injurious. Medicinal treatment should only be resorted to when the general management is not followed by satisfactory improvement or when complications arise. Excepting quinine, and preferably the salicylate, no drug antipyretics are permissible. Cod-

liver oil should be given to patients who bear it well, even if we are obliged to limit or decrease the use of other fats. Creosote should be considered only as it may influence the bronchial catarrh or certain digestive derangements, it having no direct influence upon the disease. Diarrhœa not controllable by proper diet yields best to bismuth subgallate, eight to ten grains every three or four hours, or to similar doses of bismuth salicylate every six to eight hours; moist compresses to the abdomen over night are also beneficial. Cough, with expectoration, should not be interfered with; when due to congestion it yields to rest; when dry and harassing, apomorphine, one-sixteenth of a grain, with or without codeine, one-eighth of a grain, every two or four hours, gives the best results. Moist, soothing inhalations should be tried before resorting to opiates. Night-sweats yield less rapidly but more lastingly to cold sponge-baths at bedtime, and the use of the ice-bag over the heart, with which the patient goes to sleep. If the sweating is due to a weak action of the heart, strychnine, one-twentieth of a grain hypodermically, should be given upon retiring, or a dose of brandy or rum in milk may benefit. Atropine should not be used. Pleurisy, either acute or the circumscribed dry form, requires strapping for the relief of pain, rest in bed, counter-irritation, and, if effusion is present, the proper medical or surgical treatment for it. Bloody expectoration and hemorrhage are, as a rule, the result of physical over-exertion and otherwise improper conduct of the patient. Rest in bed, control of the cough by morphine, hamamelis, and the ice-bag over the heart have given the best results. In anæmia the preparations of iron have been disappointing; they are not well tolerated and tend to constipation. In some cases the blood-conditions improve under otherwise correct management and liberal diet, together with proper out-door life. In about twenty cases the Klebs tuberculin has given highly encouraging results, and in large doses has an apparently destructive effect upon the tubercle bacilli, with subsidence of the fever and rapid improvement of physical signs and general symptoms. There are not any disagreeable or undesirable after-effects. In a general way there is nothing more important than a good stomach, a good heart, and good common-sense on the part of the patient. They are the requisites for recovery.—*Transactions of the North Carolina Medical Society*, 1894, p. 30.

The Treatment of Broncho-pneumonia.

Dr. Nothnagel states that the auxiliary muscles of respiration must be acted upon in order to avoid asphyxia, cyanosis, and carbon-dioxide-poisoning. For this purpose water-baths or Priessnitz's wet cloths are used. Under this treatment the patient is placed in a bath and a spray of cold water driven against the thorax, or the ordinary douche will serve the same purpose. To avoid venous stasis the position of the patient should be changed every hour by the nurse, and he should not be allowed to remain long upon his back. The nurse should also see that the patient makes four or five deep inspirations every half-hour. Drugs are administered for the purpose of removing the secretions by expectoration. Inhalations are useless, and ammonium chloride, sodium chloride, oleum terebinthinæ are no better. A few expectorants may suit special cases, as decoction of senega and ammonium chloride. To relieve cough we have at hand narcotics, such as belladonna, hyoscyamus,

that greatly weaken, and are avoided at the present day. Codeine, in double the dose of morphine, has been much lauded as a remedy for cough. If the cough be severe, morphine with aqua laurocerasi may be given in small quantities; the latter alone does not afford relief. Morphine may be used with benefit in some so-called dry coughs—those produced by hyperæsthesia of the vagus branches in the lung. If a patient swallows all that comes up, or when he is too feeble, and in this case moist râles will be heard, or when the mucus is tenacious, we must not conclude that the cough is dry. For tenacious secretions the stimulating expectorants, as benzoic acid and senega, are indicated, or sodium chloride with warm water as an inhalation may give relief. —*The Medical Press*, 1895, No. 2905, p. 29.

Kola.

Dr. E. B. Smith states that the great value of this drug is due to the alkaloids theobromine and caffeine, and to the principle known as kolanin. As an astringent kola has been used with marked success in cases of atonic diarrhœa, its combined astringent and tonic properties producing most satisfactory results. In the treatment of summer complaint of children it takes the place of opium, with none of its disadvantages. It aids digestion by increasing the activity of the salivary glands, augments the output of the digestive fluids, and is, therefore, beneficial in that form of dyspepsia which accompanies diarrhœa. Since it exerts a sustaining effect upon the vasomotor system, it becomes an important remedy in the treatment of children with diarrhœa when the circulation is enfeebled. When long-continued exertion is demanded, and little food is obtainable, it seems to possess sustaining properties similar to those of coca. It lessens tissue-waste, as is shown by the diminished excretion of urea. Upon the circulatory system it is a tonic stimulant; the pulse is increased in strength and frequency. It is also slightly diuretic. In alcoholism it may take the place of liquor; it builds up the nervous system, enabling the patient to withstand the craving for alcoholic stimulants. It aids in overcoming the indigestion, and is beneficial in relieving the vomiting. In phthisis it lessens the cough, and is of value. In pulmonary hemorrhage it is useful. Locally, it appears to be an astringent.—*The American Therapist*, 1895, No. 7, p. 176.

The Hypnotic Value of Trional.

Dr. G. A. Hewitt believes that the competition, struggles, anxieties, and responsibilities of commercial and professional life in populous communities constitute such a strain upon the nervous system in general, and the brain in particular, that insomnia, or a not very deep nor refreshing sleep, results. For this drug he claims that ten to twenty grains in warm fluids will produce sleep without producing toxic manifestations, depression of the action of the heart, or the force of the circulation. The sleep is sound and refreshing; the patient awakens readily and with a clear head. There is little or no tendency to the formation of a habit, as no cumulative effect has been observed in some instances when the drug has been given without sufficient interval of time to allow for thorough elimination from the system. If administered for

considerable periods of time and in large doses, it is a matter of prudence to watch the kidneys. It is useful in simple insomnia, in many chronic diseases unattended with severe pain, in mental affections of a mild character or in an early stage, and in the restlessness, excitement, and insomnia of insane patients.—*The Medical Bulletin*, 1895, No. 2, p. 41.

The Treatment of Chlorotic Dyspepsia.

Dr. Henri Huchard directs that milk shall be used, or, if this is badly borne, pure water or a hot weak infusion of tea (hot drinks excite the gastric secretion), eggs, *purée* of vegetables, lean fish, fowl, and cooked fruits. One-half hour before the meal a small dose of an alkali, as sodium bicarbonate, seven grains, should be prescribed, for the purpose of exciting the flow of gastric juice. At the same interval after it a Madeira glass of hydrochloric acid in solution in water, one to two hundred and fifty. The hydrochloric may be replaced by lactic acid, fifteen to thirty grains, after meals. It is necessary to forbid the use of wines, quinquinia wine, strong beers, alcoholic drinks, and stimulating food. If there are gaseous formations, lavage, either of pure water or water containing salicylic acid, one per mille, is indicated. After two to four weeks of this treatment the use of the preparations of iron can be begun, of which the proto-salts are preferable, oxalate, lactate, chloride, and iodide, in small doses, one and one-half grains, twice daily, and not to exceed six and one-half grains daily, if we shall avoid symptoms of gastric intolerance.—*Journal des Praticiens*, 1895, No. 3, p. 33.

The Treatment of an Obstructed Bowel.

Dr. W. Thornley Stoker has never seen the application of heat and rubefacients do substantial service. Of belladonna, if used freely and early in the disease, he holds a favorable opinion, and its best therapeutic effect is when uncombined with opium. Its chief use is in cases of peristaltic paralysis due to tympanites or fecal accumulation, but it must be given in full and repeated doses, and pressed until it shows its constitutional effects of either dilated pupil or dryness of the throat; it may be combined with calomel. Sodium sulphate is useful in hourly doses of one or two drachms, until an ounce or more has been taken. For washing out the bowel an ordinary red rubber tube, such as is used for lavage, presents the advantages that: 1, it can do no injury; 2, the fluid pressure can be regulated; 3, enormous quantities of water can be used; 4, the currents created by alternating pressure exert a solvent effect upon feces; 5, the operation can be carried on for a long time without exhausting the patient.

Dr. Julius Althaus believes that in the case of absolute mechanical occlusion, such as strangulation by bands, etc., the knife affords the only chance for the patient's recovery. Two cases are cited, in which the faradic current applied through an insulated metallic sound in the rectum and a moistened conductor to the abdominal parietes in the region of the sigmoid flexure, resulted in cure. Bondel and Laral have used the constant current as an electric injection, the rectum filled with salt water connected with the positive pole acting as an electrode; the negative pole is a large electrode upon the abdomen. The strength of the current is regulated by the

susceptibility of the patient. The pseudo-strangulation and fecal obstruction relief has appeared in from five to twenty minutes. If a real obstacle is to be overcome, the current should be reversed after five or six minutes.—*British Medical Journal*, 1895, No. 1778, p. 186.

The Treatment of Gall-stones.

Dr. F. von Oefele, noting the difficulty in administering the quantity of olive oil which is prescribed for hepatic colic, proposes butter as a substitute. After the morning spring water at Neuenahr has been taken, fasting, and a quarter-hour exercise has been finished, seven to ten drachms of fresh, unsalted butter are taken with a little white bread. Then, after another quarter-hour of exercise, comes breakfast. The butter has a favorable influence upon the constipation, and if the stools are free from or deficient in biliary coloring-matters, they now become darker. The appetite was not affected, save in two instances, when it was unfavorably influenced. In cutaneous irritation this remedy in some cases is beneficial. For gall-stones this remedy is certainly curative, but even if it does not remove them, it is useful as a symptomatic remedy.—*Therapeutische Wochenschrift*, 1894, No. 2, S. 27.

The Modern Treatment of Typhoid Fever.

Dr. Frederick C. Shattuck considers that this disease involves two things which may make more or less contradictory therapeutic demands: (1) The duration and general character of the disease, which brings in its train the almost certainty of tedious convalescence and the dangers of death from exhaustion, and (2) the constant local lesion, the intestinal ulceration, the extent and depth of which we have not means of estimating or even inferring. In regard to diet, is it not possible that we sometimes make a mistake in dieting our patients with reference to their fever rather than with reference to their digestive capacity? They may have anything that is easy of digestion, and which leaves such residue as may reasonably be expected not to excite undue peristalsis and irritation of ulcerated Peyer's patches. Eggs (raw and soft-boiled), custards, animal broths, strained gruels, ice-cream, junket, blanc-mange, even scraped or very finely minced meat, in addition to milk, have been used during the past two years without cause for regret. The range of food should be as wide as the patient can digest, and such as is not likely to prove irritating to his ulcerated intestines. Water should be given freely internally to promote the elimination of soluble poisons. There is no evidence to show that the disease can be aborted by early purgatives—calomel, for instance—followed by more or less continued antiseptic treatment. The use of intestinal antiseptics should be limited to cases in which diarrhœa, very offensive movements, meteorism, or other special indications are present. Small doses of bismuth salicylate or subgallate are preferable; the former may be combined with beta-naphthol. In cases characterized by constipation water enemata every second day are ordered—glycerin enemata cause too much peristalsis. Among antipyretics phenacetin is the safest of those in common use. Observations in hospital show no marked or constant difference in the antipyretic value of cold sponging at 60° F. for twenty

minutes, the cold-pack at 60° F. for fifteen minutes, or the full bath at 70° F. for ten to fifteen minutes. A full bath at 60° F. has a much more decided antipyretic effect. The use of alcoholic stimulants calls for the exercise of more or less judgment. In a considerable proportion of cases they are never required unless the tub-bath is used, reaction from the temporary depression, so often following this method of the application of cold water, being hastened by small quantities of spirits and water.—*Boston Medical and Surgical Journal*, 1894, vol. cxxxi. p. 604.

Dr. P. Le Gendre states that delirium at the commencement can be best treated by baths if there is no contraindication to their use. If the patient is more tolerant of warm baths, progressively cooled, these may be first tried. If he rebels against the duration of the bath, cold affusions to the head with successive lowering of the temperature may be employed. If this symptom appears in the second week, it is quite likely to be due to the elevated temperature, and reduction of this will relieve the delirium. If in spite of this, this symptom is accompanied by absolute insomnia, or there is a tendency to get out of bed, opium, chloral, or the bromides are indicated. If the condition of the heart is questionable, chloral must be used with care or even abandoned. If there is an intense albuminuria, scanty urine, or a tendency to constipation, these are reasons against the use of opium. The delirium of the third week is apt to be due to weakness, and calls for nourishing fluids, peptones in the bouillon, milk, alcohol, and generous wines. Headaches accompanied by gastro-hepatic symptoms are relieved by ipecacuanha. If associated with elevated temperature, compresses of vinegar, ether, or cherry-laurel water upon the forehead, or cold affusions to the head and neck during the bath, or, if there exist symptoms of cerebral congestion, a constant application of an ice-bag is indicated. Sometimes an ointment of potassium cyanide, 0.10 to 0.20 in cold cream, 20, upon the forehead and temples is useful. Insomnia in the first week is usual, and generally yields to bathing. If it is persistent in the second or third week, the cause should be ascertained—uncomfortable bed, lack of ventilation, too high temperature of the room, or bad odors. If this fails, opium, and especially associated with camphor, is useful. This may be associated with the bromides, or tincture of cannabis indica may be tried. Quinine in sufficient dose in the evening may be a hypnotic.—*Bulletin Général de la Thérapeutique*, 1894, 44e liv., p. 461.

A Plea for Venesection.

Dr. George C. Laws thinks that the rule is universal that in all cases of inflammation so serious from degree or position as to involve danger to life, bleeding should be employed in the early stages, unless forbidden by general debility or the low grade of the fever.—*The Therapeutic Gazette*, 1894, No. 12, p. 806.

Treatment of Gonorrhœa.

Dr. Pellissier reports that, basing his observations upon the energetic action of citric acid upon the bacillus of diphtheria, and knowing that the gonococcus is best cultivated in an alkaline medium, he has used six

injections daily of this remedy in simple water (1 to 100), cure resulting on the eighth day. As irrigations (8 to 1000), used once daily, this drug has cured in about eight days.—*Bulletin Général de la Thérapeutique*, 1894, 46 liv. p. 520.

MEDICINE.

UNDER THE CHARGE OF

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Indurative Mediastino-pericarditis.

Harris (*Medical Chronicle*, November, December, 1894, and January, 1895) has collected all the cases of this interesting lesion (which was first carefully described by Kussmaul), and records three cases.

Post-mortem two varieties of the affection can be distinguished. One with adherent pericardium and marked increase of the fibrous tissue in the mediastinum. The exterior of the pericardium is closely adherent to the surrounding parts. This is the indurative mediastino-pericarditis. In the other variety there is an adherent pericardium with thickening of the sac, adhesion to the surrounding parts, but little or no general mediastinitis.

There are in addition rare instances, forming a third group, in which, without pericarditis, there is an increase in the fibrous tissues of the mediastinum—a chronic mediastinitis.

Of the twenty-two cases which he has collected, nine occurred in persons under eighteen years of age, only two in persons over thirty; seventeen cases were in males, and five in females. Usually there was a history of some acute illness, generally pericarditis. Some of the cases are tuberculous.

The symptoms indicative of the indurative mediastino-pericarditis develop at a late period. In many instances the onset is very insidious.

The symptoms have been chiefly dyspnœa, venous engorgement, cyanosis, cardiac enlargement, increase in the size of the liver, and general dropsy or ascites, with the pulsus paradoxus and inspiratory swelling of the vessels of the neck. In some cases the posture has been suggestive, as Jaccoud mentions: the patient sits up in bed with the trunk bent forward; but this, after all, is not an uncommon position in cases of pericarditis with effusion.

The duration of the cases varies. It is often difficult to say when the affection began. The period from the onset of symptoms which may be attributed to the affection to the fatal termination varies from a few months to several years. The cause of death is usually a gradual cardiac dilatation, or, in the tuberculous cases, an acute general infection.

The ascites which occurs in these cases, and which may be unaccompanied

by general anasarca, is due sometimes to a peritonitis with chronic capsulitis and deformity of the liver.

A very interesting point in Harris's paper is a careful discussion of the pulsus paradoxus, in which the beats at the wrist become smaller during the act of inspiration. It is, of course, not a peculiarity of a mediastino-pericariditis, and is seen under many conditions. Kussmaul thought it was due to a dragging of the adhesions external to the pericardium on the aorta; but there are many observations of the occurrence of the paradoxical pulse where there were no bands of adhesion in the chest, so that it is evident that other agents are capable of producing it. Harris concludes that "cardiac weakness from many causes appears to have been a not infrequent condition present when a pulsus paradoxus was present, and it is quite probable that such heart-weakness, in some way which at present is not clear to us, contributes to, if it is not the actual cause of, this peculiar form of pulse in some of the cases of mediastino-pericarditis where the arrangement of the adhesions is not such as will account for a narrowing of the aorta or main arteries in the thorax."

Primary Cancer of the Duodenum.

Pic (*Revue de Médecine*, January, 1895), after a long and minute study of primary cancer of the duodenum, draws the following conclusions:

I. Primary cancer of the duodenum, in the great majority of cases, like primary cancer of the intestine in general, has an annular form. On this account it is very apt to produce stenosis of the digestive tract.

II. The symptoms of this cancerous stenosis vary according to the height at which it is produced.

A cancer above the ampulla of Vater has a symptomatology almost identical with that of cancer of the pylorus.

A cancer below the ampulla presents, besides symptoms of malignant pyloric stenosis, signs indicating a permanent reflex of the bile and the pancreatic juice into the stomach.

A cancer round the ampulla presents, according to the case, a symptomatology more or less resembling the one or the other of the first two forms; it is a composite form.

III. Among the so-called cancers of the ampulla of Vater we may place:

1. Cancers of the duodenum in which the ampulla is invaded; nothing distinguishes these tumors clinically from ordinary duodenal cancers; they are included in our third form.

2. Cancers primary in the head of the pancreas.

3. There exist some cancers appearing to be developed at the expense of the ampulla, and presenting the clinical picture of icterus from retention—a picture comparable to that of cancer of the head of the pancreas. These cancers must be distinguished clinically and histologically from cancers of the duodenum strictly speaking, and must be classed with pancreatic cancer, of which they appear to constitute aberrant varieties (glandular or excretory).

4. *a.* In most of the cases there is a contraction at the site of the tumor and a dilatation toward the stomach, tending to involve this organ and frequently accompanied by pyloric insufficiency.

b. The histological examination has revealed up to the present, amongst the primary tumors of the duodenum, cancers of the investing epithelium type (cylindrical epithelioma), of the glandular type (primary epithelioma of Brunner's gland), of the embryonic-tissue type (sarcoma), and of the lymphatic type (lymphadenoma).

As a matter of fact, there are forms of cells entering into the constitution of the duodenum other than those found in these forms of cancer.

5. The difficulty of diagnosis in cancer of the duodenum varies according to the situation of the neoplasm; whilst the diagnosis of the form which occurs above the ampulla is ordinarily impossible, that of the two other forms is sometimes possible. Speaking in a general way, the chief differentiation is to be made between duodenal cancer and stenoses of the digestive tube of supra-duodenal (pyloric) or infra-duodenal origin; and between duodenal stenoses of extrinsic origin (from compression).

6. The topographical diagnosis is of greatest importance from an operative point of view, which will differ according to the situation of the neoplasm in the duodenum. When the diagnosis is impossible early exploratory laparotomy will be necessary, and in most cases will be only palliative.

Pathogenesis of Gastric Ulcer.

A good review of the recent theories of the pathogenesis of gastric ulcer is given by Grieg (*Canadian Practitioner*, February, 1895).

According to this author, the exciting cause is essentially peptic in character, some predisposing condition of the gastric mucous membrane being necessary to allow of the digestion of the affected area; and it is these predisposing causes that Grieg discusses.

Trauma *per se* would seem insufficient to cause a chronic ulcer, but occurring in a poorly nourished, anæmic person, the author seems to think that ulcer might result.

Embolus, except in very rare cases, may be practically excluded, as no convincing case of ulceration following embolus is on record, and the ulcers produced experimentally by emboli are multiple and occur in the fundus, differing entirely from the solitary pyloric ulcers of the human being.

Thrombosis and various degenerative changes in the bloodvessels would seem to be the most likely predisposing causes, several cases having been reported in which the ulceration was associated with atheromatous and fatty changes in the arteries, or with endarteritis obliterans.

Hyperacidity, the author states, is a frequent, but not invariable, accompaniment of ulcer. Its presence, he thinks, is just as likely to be the result as the cause of the disease, though it would undoubtedly retard healing.

Ulceration of the Duodenum in Bright's Disease.

In analyzing nearly eighteen thousand autopsies, with special reference to duodenal disease, Perry and Shaw (*Guy's Hospital Reports* for 1893) found twelve in which duodenal ulcer coexisted with Bright's disease, and to these twelve cases added four from outside sources.

Among these sixteen cases one was at once distinguishable from the rest

on account of the apparently indisputable relation between the nephritis and the ulcerative process. This case was that of a man, aged thirty-six years, admitted, after a two months' illness, for albuminuria and diarrhœa, blood having been present in the stools. The patient died after three days' residence in the hospital; the kidneys at autopsy showed marked cirrhotic changes, and the left ventricle was hypertrophied. Ulcerated areas such as are found in Bright's disease were present all through the large and small intestines, and the lower part of the duodenum was involved in the process.

The case seems to prove that the ulcerative entero-colitis of Bright's disease may rarely extend as high as the duodenum.

In the other cases the type of ulceration did not materially differ from that found when Bright's disease is not present. Of the twelve original cases the nephritis was seven times of the interstitial variety, four times of the tubal, and once of the two combined.

According to the authors, about 7 per cent. of persons die from nephritis, and as over 15 per cent. of their cases of duodenal ulcer show an associated nephritis, they conclude that a causal relation exists between the two.

Tuberculous Meningitis with Recovery.

The question of recovery in tuberculous meningitis has hitherto been obscured by doubt as to the diagnosis. This was not so in a case reported by Freyhan (*Deutsche medicinische Wochenschrift*, 1894, No. 36). Freyhan made a diagnostic puncture of the spinal canal in the lumbar region. About sixty cubic centimetres of slightly turbid, pale, serous fluid spurted out. In the sediment pus corpuscles and tubercle bacilli were found. The patient improved rapidly, and in three weeks after the puncture was able to leave the bed. Recovery can therefore not be looked on as a reliable sign in distinguishing between various forms of meningitis. Incidentally the case raises the question of the therapeutic value of puncture in meningitis.

Paralysis Consecutive to a Pseudo-membranous Angina Recognized as Non-diphtheritic by the Bacteriological Examination.

Under the above title Bourges (*Archives expérimentelle de Pathologie*, January, 1895) describes a case with the clinical picture of diphtheritic paralysis, but in the throat of which no Klebs-Löffler bacilli were found. The case occurred in a boy of seven years, in whom appeared suddenly high fever, headache, and pain in the throat. The next day the tonsils were gray and swollen, but no false membrane was present. Two days later the local signs had gone. Nine days from the first attack a second attack of malaise and fever occurred, with marked erythema on the neck, forearms, and limbs, but not on the trunk; the throat was normal, and the eruption disappeared two days later without desquamation.

A week from the beginning of this second attack the patient was taken with headache, fever, and sore-throat, and the next day a white, easily detachable false membrane was present over the tonsils; this remained until seventeen days later, when it had entirely gone, and the child was in fair health, though remaining pale and thin.

Sixteen days after the disappearance of the membrane the child became restless at night, and the next day complained of abdominal pain and headache; a little fever was present.

Two days later convergent strabismus developed, followed in forty-eight hours by nasal intonation of the voice and regurgitation of liquid food.

The regurgitation disappeared in a short time, but three days after its appearance the child was unable to stand, though still able to move its limbs in bed.

The paralysis remained incomplete, sensation normal, knee-jerks absent. Cure was complete at the end of two months.

The bacteriological examination of the false membrane showed the only virulent organism present to be the streptococcus pyogenes. The child's mother, who had been constantly nursing him, was attacked during his illness by a similar pseudo-membranous angina in which the sole pathogenic organism was the streptococcus pyogenes.

Septic Diphtheria and Secondary Infection in Diphtheria.

It is often supposed that septic diphtheria, characterized by a severe local lesion and very severe general symptoms, is due to mixed infection with streptococci. Genersisch (*Jahrbuch für Kinderheilkunde*, Bd. xxxviii.) examined the internal organs and the blood of twenty-five cases of this kind, soon after death, in order to discover the actual conditions. He found that mixed infection did not necessarily accompany the clinical picture of septic diphtheria. Cases with the most pronounced septic symptoms were met with in which streptococci were not found in the internal organs; while, on the other hand, those organisms were found in cases without septic symptoms. The author, therefore, believes that the diphtheria bacilli alone are able to produce the so-called septic symptoms. The streptococcous infection he thinks can be discovered during life by examination of the blood.

Reiche (*Centralblatt für innere Med.*, 1895, No. 3) has examined the question of mixed infection in diphtheria in forty-two fatal cases treated in the Hamburg-Eppendorf Hospital. In all cases the diagnosis was established by the finding of Löffler's bacillus in the local lesion, and in all various other bacteria were found. These cases varied much in the extent of the local process and the number and severity of the complications. Twenty-seven required tracheotomy. In 64.3 per cent. of the cases both streptococci and staphylococci were found in the organs; in 45.2 per cent. streptococci only. Diphtheria bacilli were found twice. A very interesting fact was the finding of streptococci in the case of a child who died on the second day of the disease. In two other cases secondary infection was present by the third and fourth day. The possibility of the early occurrence of this important complication the author rightly looks on as an important limitation of the value of the antitoxic treatment as used at present.

Digestion-leucocytosis in Ulcer and Cancer of the Stomach.

Schneyer has reached some interesting conclusions in regard to the proportion of leucocytes during digestion in the gastric diseases mentioned

(*Internationale klin. Rundschau*, 1894, No. 39). In all of eighteen cases of carcinoma there was constant absence of the usual leucocytosis after meals. In three cases of benign stenosis and seven of simple ulcer the leucocytosis promptly appeared. A case of chronic ulcer with marasmus showed no leucocytosis. Schneyer explains the non-appearance of leucocytosis in cancer by disturbances of absorption, of peptonization, and of the lymphatic apparatus, the latter being rather hypothetical. The practical application of the observations is that the absence of digestion-leucocytosis does not positively indicate carcinoma, but that its appearance speaks against that disease.

Vicarious Menstruation through the Lungs and its Relation to Tuberculosis, with Remarks on the Treatment of Pulmonary Hemorrhage.

Kober (*Berliner klinische Wochenschrift*, 1895, No. 2) describes the case of a girl of nineteen years, with a tuberculous family history, who had severe hemorrhage from the lung at the menstrual period, without previous signs of pulmonary disease. The menses proper lasted only a few hours, but the pulmonary bleeding continued for three days with great severity. The bleeding then subsided, after nausea and vomiting had come on, and these latter symptoms lasted some days longer. During this time signs of pneumonia appeared in both bases, with mild general symptoms. In about ten days the patient seemed perfectly well. At the next period a similar set of phenomena appeared, but recovery was not complete. The patient was treated for tuberculosis, although bacilli could at no time be found. Efforts were made at the same time to bring on uterine hemorrhage at the next menstrual period, but in vain, and a third time pulmonary hemorrhage appeared, the amount of blood being less than at the former periods. Vomiting, fever, and a fresh pneumonic attack followed. The cough remained, diarrhœa appeared, loss of flesh and strength, with night-sweats and high temperature varied the picture, and in four months from the first hemorrhage the patient died. Tubercle bacilli could not be found up to the last.

Kober thinks the patient had tuberculosis at the time of the first hemorrhage, and that this was the cause of the vicarious menstruation, the lung forming a *locus minoris resistentiæ* which suffers in the high arterial tension of menstruation.

An interesting fact is that no remedy seemed to check the hemorrhage, but that each of the three attacks was cut short by nausea and vomiting. This recalls the treatment of Graves and Trousseau, the former using ipecac in nauseating, the latter in emetic doses. Kober proposes a combination, first causing vomiting, and then keeping up nausea by small doses of the remedy. The cause of the natural cure Kober does not explain, but suggests irritation of the vomiting-centre by lack of oxygen from loss of blood, or in a reflex way by the blood in the alveoli.

Acute Renal Degeneration due to Convulsions.

Reiche reports some cases in which necrosis of the renal tubules seemed to be due to repeated convulsive attacks. For the interesting anatomical

and clinical details the original must be consulted (*Centralblatt für innere Medicin*, 1895, No. 2). Reiche thinks the degeneration is due to intoxication by the products of muscle-fatigue. That these substances are poisonous is well known, and that the renal epithelium is extremely sensitive to such poisons is equally familiar. It is possible that the cases of albuminuria observed after marching or mountain-climbing, and those which occur after severe epileptic and other convulsive attacks, represent mild forms of a similar process. The author holds that delirium tremens and other forms of delirium, with restlessness and motor excitation, are possible causes of this form of renal degeneration. If this be so, the beneficial effect of hypnotics in delirium tremens, and of narcotics and other sedatives in uræmic and other convulsions, becomes clear, since by their help the organism is spared a dangerous complication. Animal experiments, and experiments with the toxicity of the urine and blood of patients with severe convulsions, may add much to our knowledge of this subject.

SURGERY.

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A Case of Prostatic Hypertrophy Cured by Castration.

LÜTKENS, of Cella, reports the following case in the *Deutsche medicinische Wochenschrift*, No. 5, 1895: A man, aged sixty-three years, sought advice on account of an obstruction in passing urine which necessitated the frequent use of the catheter. The patient had experienced trouble in urinating for several years, but he did not seek medical advice until the use of the catheter became difficult and attended with severe pain. Frequently clotted blood came out with the instrument. When the patient consulted Lütkens he could not pass urine spontaneously. On digital examination the prostate was found to be as large as a hen's egg, and projected into the rectum. The condition was so far advanced that the author decided that some radical measure was necessary. Castration and ligation of the internal iliac arteries were considered, and the former decided upon, because he had not seen the operation of ligation of the internal iliacs and he had no opportunity to practise it upon the cadaver; the technique is complicated and difficult, and

besides, in gynecological practice, the results of castration for uterine myomata are better than follow ligation of the uterine arteries.

Double castration was performed on October 10th. Ten days after the operation there was a positive decrease in the size of the left half of the prostate; on November 6th (twenty-seven days) this half of the gland could not be felt, but the right lobe was still as large as a small walnut. On March 4th an examination proved that all signs of hypertrophy had disappeared. The following table shows the rapidity of the restoration of the normal function of urination:

	The patient passed in 24 hours	Drawn with catheter.
October 1–10	. . . . .	2000–2500 c.cm.
" 10	Operation.	
" 11	. . . . .	1900 "
" 12	. . . . .	2100 "
" 13	. . . . 400 c.cm.	1750 "
" 14	. . . . 500 "	2000 "
" 15	. . . . 500 "	1650 "
" 16	. . . . 750 "	1500 "
" 17	. . . . 900 "	1500 "
" 18	. . . . 1000 "	900 "
" 19	. . . . 1150 "	900 "

After October 20th the catheter was no longer needed; the patient was urinating spontaneously and without trouble. On March 4th the patient was seen, and was entirely well. No living spermatozoa could be found in the testicles after removal. At the time this seemed an unfavorable sign, as Hegar advises against removal of the ovaries after the menopause. Lütkens believes that the atrophy of the prostate which follows castration is due to the change of nutrition caused by severing the vasomotor connection which normally exists between these glands.

Gonorrhœal Immunity.

Jamin (*Ann. des mal. des org. Genito-urin*, November, 1894), in reviewing a recent work by Guiard on gonorrhœa, comments favorably on the author's theory regarding inoculation and immunity, based on the following facts:

1. Gonorrhœa is a self-limited disease, recovery following after a varying length of time, without any treatment other than ptisanes, baths, and hygiene. 2. Abortive treatment does not succeed rapidly and fully until the period of acute inflammation has passed. 3. A patient, either male or female, apparently, though not really, cured is capable of transmitting a subacute gonorrhœa—that is, the colorless drop of secretion is capable of inoculation if it contains a few gonococci. The author consequently believes that the gonococcus during its evolution modifies its culture-media—*i. e.*, the urethral mucous membrane.

It continues to grow there but becomes more and more attenuated and latent, until it finally disappears. Unless this is true, a gonorrhœa untreated would last indefinitely, the cocci multiplying and reinoculating themselves in the infected urethra continuously. It is at the time when the virulence

begins to diminish that abortive treatment succeeds. If, on the other hand, this attenuated gonococcus comes in contact with a healthy mucous membrane in another individual it is immediately rejuvenated, prospers, and produces acute gonorrhœal symptoms, since it finds there a suitable culture-media, which is not exhausted with that from whence it came and where it was about to die.

The objectors to this theory say: "If you admit that the *rôle* of the culture. media is thus superior to that of the microbe, how can you explain acute attacks in men having gleet? How can their exhausted mucous membrane become reinoculated?"

Guiard believes this reinfection is impossible in cases where the gleety discharge contains cocci. He, however, reserves his opinion regarding this form of relative immunity until further facts are procured.

The Results of the Injection of Living and Dead Bacteria into the Pelvis of the Kidney.

Schnitzler and Savor (*Fortschritte der Med.*, December, 1894) conclude, from a long series of interesting experiments on dogs, that we should not speak of pyogenic and non-pyogenic bacteria, but that we are only justified in saying that certain bacteria have, under certain circumstances, the power to produce suppuration. In regard to the injection of dead bacteria, especially proteus, it is noteworthy that macroscopic suppuration in the nephritic tissues was not observed. When a round-celled infiltration was present still the running together and necrosis of tissues were absent. A quantitative and not qualitative difference could be made out.

The most marked result of their research is the fact that unwounded epithelium—*i. e.*, of the pelvis, of the kidney, and of the ureters—is no barrier against the working of dead micro-organisms; for their experiments show that dead bacteria injected into the pelvis of the kidney can produce a diffuse inflammatory change in the interstitial tissues of the entire kidney.

Early Symptoms and Diagnosis of Tuberculous Arthritis.

Judson (*Revue d'Ortho.*, November, 1894) says that we should never forget that "the pain of coxalgia is felt in the knee and that of Pott's disease in the stomach." In coxalgia this symptom is, however, subjective, and is not so valuable as the objective signs that are more marked. Amongst the first symptoms he places partial impotence in the limb, which disappears and reappears again later. It shows itself in the morning when the patient arises, and then disappears. The rhythm in walking is broken, the sound foot remaining longer down and coming down in a peculiar manner, as if to keep the weight off the diseased joint. The attitude in repose is also marked; the weight is sustained by the sound limb, the other being held in advance and to one side. To this is joined a muscular atrophy, with flattening of the buttock, with a marked shortening and shallowness of the gluteal fold. Measurement will also show a decrease in size of the entire limb. Reflex muscular obstruction of passive movements is a most marked early symptom, met most frequently, perhaps, in movements of rotation. Seat the patient on

a table with the legs hanging down; make them swing with a lateral motion, and it will be noticed that the arc described by the diseased limb is less than the other. Now place the patient upon a hard bed or table and rotate the limbs inward and then outward. On the sound side the external and internal borders of the foot will touch, or nearly touch, the table, while on the suspected side rotation will be limited.

The limitation of adduction, abduction, and flexion can be studied by passive movements on both limbs. The patient may test this for you: ask him to kiss his knee—it is easy on the sound side, impossible on the other. These examinations should be made with care and gentleness, for our purpose is to study the slight differences in muscular reaction and to recognize the repugnance of the muscles to relaxation and the allowing of certain movements which their spasmodic action forbids.

As the disease advances these signs lose their importance, and are masked by three which, when present and recognized, make the diagnosis evident: 1. The enfeeblement of the patient is constant. 2. The atrophy of the muscles is very marked, due in part to the excessive activity of one limb and the rest of the other, inactive. 3. The total, or nearly total, absence of motion in the joint.

The Presence of Albumin in the Urine after Etherization.

The prevalent opinion that albumin is found in the urine after ether-narcosis, and that this is consequently an objection to its use, has been, to a certain extent, disproved by the researches of Barensfeld (*Münch. med. Woch.*, 1894, No. 41). He examined the urine of one hundred and fifty patients before and after either-narcosis and found albumin present in only four, and in three of these had no bearing on the ether-narcosis. In one, therefore, out of one hundred and fifty cases albumin was found in a small amount after ether, but disappeared after five days. The author says, therefore, that a true ether-nephritis does not exist, and agrees with Fueter and Roux in saying that albumin cannot be found after etherization where it could not have been found previously. He also relates a case of hydronephrosis in a three and one-half year old child in whose urine no albumin was found after etherization and nephrectomy.

Surgical Treatment of Carcinoma of the Stomach.

Klemm (*St. Peter. med. Woch.*, 1894, No. 49) summarizes his opinions on this subject as follows:

1. The therapeutic treatment of these tumors differs in no respect from that of cancer in any other portion of the body. It is purely surgical.
2. Operation is to be recommended, when possible, before the tumor is palpable.
3. Examination should only be made under deep narcosis.
4. Exploratory incision will in many cases give definite conclusions.
5. Those cases only should be recommended for resection of the pylorus where the tumor is freely movable and there is no metastatic involvement.
6. If these conditions are not present, the formation of a fistula between the stomach and jejunum is indicated.

7. This operation should not be delayed until the patient is nearly dead of hunger, and the knife then used as a last resource.

8. Under such circumstances a greater mortality is to be expected than from the simple operation.

An Addition to the Knowledge of Tumors of the Penis.

Buday (*Arch. für klin. Chir.*, 1894, Band 49, Heft 1) describes two cases of malignant growths of the penis, with the macroscopical and microscopical examination. The first case showed a cystic adenopapillary structure of undoubted malignancy, which was without doubt a true epithelial carcinoma arising from the inner layer of the prepuce. The second, however, differed both in clinical and anatomical characteristics from ordinary epithelioma, and was more like an elephantiasis of the penis. The thickening of the entire penis, with no involvement of pulmonary structures, made this seem probable, while the rapidity of the growth, paraphimosis, and the resultant swelling of the penis and formation of urinary fistulæ strengthened this probability. The hardness of the tumor and its sharp, deep outline, the formation of the fistulæ without inflammation, and the discharge of comedo-like material, and not pus, were, however, evidence against this diagnosis. The microscopic examination showed the tumor to be made up of a papillomatous epithelioma, the papilloma arising from an atypical development of tissue-hypertrophy. The origin of this tumor was probably from the mucous membrane of the fossa navicularis.

OTOLOGY.

UNDER THE CHARGE OF

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Congenital Fistula of the Auricle.

Dr. Vauthier observed a congenital fistula in the anterior superior portion of each auricle, giving exit to a slight, yellowish, somewhat malodorous liquid, in a female child born prematurely between the seventh and eighth month of gestation. The external ear was otherwise healthy. There were seven other instances of this peculiarity in the same family.—*Annales de la Policlinique de Paris*, vol. iii., No. 3.

Tuberculous Perichondritis of the Auricles.

Haug, of Munich, has seen four cases of perichondritis in tuberculous patients in addition to external signs of disintegration of the affected parts, infiltration of lymphatic glands, and a tendency to ulceration. The diagnosis was confirmed by the microscope. Treatment by generous opening, excision

of glands, and dressing with Peruvian balsam and iodoform. Prognosis is favorable, notwithstanding deformities of auricle.—*Langenbeck's Archiv*, vol. xliii.

Removal of an Exostosis of the External Auditory Canal.

Hecke has observed a patient, twenty years old, with exostosis occupying nearly all the external auditory canal. This developed during otitis media suppurativa, induced retention of pus in the drum-cavity, and brought about destruction of the tegmen tympani, exposing the dura mater. There was also a fistula in the mastoid apophysis. After detachment of the auricle the drum-cavity was exposed and the mastoid antrum was opened by following the fistula. Cure in six months by formation of epidermis in the vast cavity.—*Annales des Maladies de l'Oreille*, vol. xx.

Epithelioma of Auricle.

Dench regards epithelioma of the auricle as comparatively rare. He cites twelve cases of others, and reports a case where a hard tumor originated at the base of the tragus and extended slightly on the lower posterior border of the meatus. Ablation two months after first appearance was followed by complete cure. The prognosis seems more favorable here than anywhere else in the body.—*Archives of Otology*, vol. xxii.

Bromide of Ethyl in Oto-laryngology.

So far as concerns otology, Helme thinks, in children who resist all manipulations of their ears, this drug would do well. Under anæsthesia from it a polypus could be extracted, a foreign body removed, a Wilde's incision made, or a curetting of the cavum tympani performed (if it ever should be done.—Rep.); also the Eustachian tube could be catheterized while patient is under the effects of bromide of ethyl. The author agrees with Hartmann and Bourbon, that for short operations nothing superior as an anæsthetic is known than bromide of ethyl.—*Annales des Maladies de l'Oreille*, vol. xx.

The Treatment of Chronic Suppuration of the Middle Ear by Excision of the Auditory Ossicles.

Dr. William Milligan, of Manchester, England (*British Medical Journal*, No. 1706), demonstrates the uselessness of all local treatment in many cases of chronic suppuration of the middle ear as long as necrotic ossicula and obstructing synechiæ remain in the drum-cavity; therefore, there is no hope of curing these cases until the necrotic ossicula are excised, the patient being under the influence of a general anæsthetic. Local treatment after this operation in the now better drained cavity is attended by good results. (Assuredly a mastoid opening for the purpose of better drainage and cure of chronic suppuration in the drum-cavity should not be thought of until excision of the diseased remnants of the membrana tympani and ossicula has been performed.—Rep.)

Essential Neurasthenia Associated with Secondary Aural and Rhino-pharyngeal Maladies.

Cozzolino endeavors to show that there is a clinical form of oto-neurasthenia connected exclusively with the labyrinth-expansions of the nerve, and can arise from the nerve-trunk. This form is often bilateral, and there is often a hyperacousma; it may be developed during cerebro spinal neurasthenia. When an individual with normal hearing complains of auditory disturbances, an examination of the cerebro-spinal axis and muscular systems should be made. Hallucinations of hearing are possible only in neurasthenics. These forms of neurasthenia accompany disturbances of sensibility and motility of the rhino-pharyngeal cavity.—Eleventh International Medical Congress, 1894. *Annales des Maladies de l'Oreille*, Tome xx.

Influence of Malaria on Diseases of the Ear.

Garzia, Naples, believes that malaria may excite inflammations of the middle ear and spread thence to the labyrinth. These affections are curable by remedies for malaria and by local means.—Eleventh International Congress, 1894. *Annales des Maladies de l'Oreille*, Tome xx, No. 5, p. 511.

Propagation of Affections of the Tympanum to the Brain by the Way of the Carotid Canal.

Koerner demonstrates anew the fact that the canaliculi conveying nutrient vessels from the carotid canal to the mucous membrane of the drum-cavity may become the passage-way of microbes of otorrhœa either with or without caries, and cause arteritis, lymphangitis, phlebitis of the venous plexuses, with participation of the lateral sinus, and even thrombosis of the carotid artery and consecutive cerebral embolism. He cites cases in which the external tunic of the artery was found infiltrated with tuberculous granulations, and admits the possibility of tuberculous meningitis having its origin in this way in bacillary otitis media.—*Archives of Otology*, vol. xxii.

Treatment of Cholesteatoma of the Middle Ear.

Reinhard states that while authors do not agree upon the etiology and anatomy of cholesteatoma, they, however, are in accord as to its prognosis and treatment, viz.: that cholesteatoma of the ear is a dangerous and even fatal malady, and the treatment consists in the entire removal of epidermic masses. In light forms of the disease, without caries of the walls of the attic, the ossicles should be extracted through the external auditory canal; and in more severe forms, complicated with caries of the tympanic cavity, this ablation should take place by Stake's method. Of cases of extensive cholesteatoma it is necessary to make one cavity of the middle ear, the antrum, and the mastoid cells, maintaining as long as possible this opening, and endeavoring to make skin-graftings into it. Of nineteen cases, a permanent opening was thus maintained in fifteen cases. When such an opening did not exist the formation of cholesteatomatous masses and suppuration continued.

Politzer, in the discussion of this communication, called attention to the fact that such a proceeding favored the propagation of epidermis in the drum-cavity, which propagation, as is well known, might be regarded as a cause of cholesteatoma.

Dundas Grant stated, at the same time, that it is of the first importance to prevent moisture after this operation, and to have recourse to the insufflation of antiseptic powders.—Eleventh International Medical Congress, Rome, April, 1894. *Annales des Maladies de l'Oreille*, Tome xx.

Surgical Intervention in Septico-pyæmia Consecutive to Otitis Media Suppurativa.

H. Nimier contributes a good paper on general surgical treatment of sinus-thrombosis and resultant pyæmia. How many such cases would never occur if the ear had not been neglected originally! Had the patient been sent promptly to an aurist, who alone is capable of treating the acute disease, the necessity to call in the general surgeon would never have arisen.—*Annales des Maladies de l'Oreille*, Tome xx.

Percussion of the Mastoid Process in a Case of Pyæmia Consecutive to an Acute Affection of that Process.

Wild advocates the percussion of the mastoid with a metallic hammer as a means of early diagnosis of caries of the temporal bone. Recently he observed a case of otitis media suppurativa in which the dull percussion sign was the only local symptom of osseous disease, which furthermore was complicated by the usual symptoms of pyæmia. Opening of the mastoid was followed by quick improvement and soon entire cure.

Moos thinks percussion of the mastoid has only a positive value. When dulness on percussion is absent we may not conclude there is no affection of the bone.—*Annales des Maladies de l'Oreille*, Tome xx.

Use of the Curette in Mastoid Operations.

C. J. Blake, after opening the antrum by a trephine or enlarging an opening already present, uses curettes of various sizes for removing diseased tissues. He employs no sutures, but applies a dry dressing. The operation is preceded by a large incision in the membrana tympani.—Eleventh International Medical Congress, 1894. *Annales des Maladies de l'Oreille*, Tome xx., No. 5, p. 511.

Association of Hysteria with Lesions of the Ear.

Gradenigo maintains that hysteria of the ear is frequent. In a hysterical subject a slight affection of the ear, either inflammatory or traumatic, causes local and general nervous phenomena disproportioned to the lesion. Most cases of otalgia in young women, when dental caries, sore-throat, and syphilis can be excluded, are due to hysteria. He cites cases of periodical hemorrhages from the ear and of hysterogenous zones of the ear. Also he states that hysterical subjects simulate deafness.—*Annales des Maladies de l'Oreille*, Tome xx., No. 5.

Pathogeny of Otitis Interna.

Gradenigo asserts that there are two forms of otitis interna: one due to the propagation of an otitis media catarrhalis, and the other to a primary disease in the internal ear, as syphilis, acquired or hereditary, parotitis, and influenza. But even in these cases there exist previously slight lesions in the middle ear, which explain the localization of the disease in the internal ear, and its gravity. Also catarrh of the nasopharynx will be found in addition to these lesions in the middle ear. Hereditary predisposition also plays an important part, so that this, with some old lesion in the ear, plus the acute or chronic infection, like influenza, diphtheria, acquired or inherited syphilis and tuberculosis, determines the occurrence of an otitis interna. The part played by syphilis is especially important, and a number of cases of supposed sclerosis are really lesions of attenuated and delayed hereditary syphilis.—*Annales des Maladies de l' Oreille*, Tome xx., No. 5.

Lesions in the Labyrinth in a Case of Leukæmic Deafness.

Wagenhäuser gives an account of a woman, thirty-five years old, attacked with leukæmia in April, and six months later with total deafness, at the time of a profound infiltration in the right arm and thigh, apparently due to interstitial hemorrhages. No data as to vertigo or tinnitus. Sudden death eight months later.

Post-mortem examination revealed normal tympana; but in the internal ears, cochlea, vestibule, and semicircular canals, extensive and copious extravasation, with numerous round cells; these lesions most marked in the semicircular canals. The primary lesion in this case had been hemorrhage into both labyrinths, productive finally in the contiguous area of an inflammatory process resulting in new formations of connective tissue and bone.—*Annales des Maladies de l' Oreille*, Tome xx., No. 5.

Deafness Consecutive to Cerebro-spinal Meningitis.

This is represented by 4 per cent. of all cases of so-called nervous deafness. When this deafness persists after the early days of convalescence the prognosis is unfavorable. There is a form which disappears spontaneously as soon as convalescence sets in. These spontaneous cures may be due to the treatment, viz.: to the action of pilocarpin.—*Annales des Maladies de l' Oreille*, Tome xx.

Kindergartens for Deaf-mutes.

Flatan (Berlin) has founded a kindergarten with the view of developing physically deaf-mutes under the age of eight years. No instruction is given in speech. Search might be made among the pupils for those still retaining some hearing, and, if such is found, hearing could be cultivated by proper exercises. Flatan thinks that such schools, supervised by a physician at once a hygienist and aurist, could in the future furnish valuable material for the scientific study of deaf-dumbness.

Politzer, in the discussion, stated that sometimes the improvement in

hearing following exercises of this function should be attributed to the perfecting of the tactile sensibility of the skin of the auricle, in consequence of which deaf-mutes are able to distinguish the different vowels pronounced near them.—Eleventh International Medical Congress, 1894. *Annales des Maladies de l'Oreille.*, Tome XX., No. 5.

DISEASES OF THE LARYNX AND CONTIGUOUS STRUCTURES.

UNDER THE CHARGE OF

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The Laryngological Society of London.

We would like to call attention to the excellent work being done by the Laryngological Society of London, and would advise all special workers in laryngology and cognate branches to secure copies of its Proceedings, which are published at short intervals. The brief discussions on cases presented to the Society often afford invaluable instruction in differential diagnosis elsewhere unattainable, while the general worth of the subject-matter is not exceeded in the transactions of any other organization.

Morbid Growths in the Nasal Passages.

An instance of pedunculated papilloma of the septum narium of the left side, in conjunction with a rodent ulcer on the right side of the nose, was shown by Mr. P. de Santi to the Laryngological Society of London, November 14, 1894 (*Proceedings of the Lar. Soc. of London*). The patient was a man, eighty-two years of age. The tumor was apparently of some five years' development. The rodent ulcer, the size of a Spanish nut, was of little more than one year's duration.

Technique for Extirpation of Malignant Disease by Thyrotomy.

In the concluding portion (*The Lancet*, 1894, No. 3722) of an article by Dr. Felix Semon, on "The Results of Radical Operation for Malignant Disease of the Larynx" from the experiences of private practice, the improved technique is described under which satisfactory results are to be anticipated by the method of splitting the larynx in front and then removing the soft parts, and likewise, if necessary, resecting some portions of the cartilages.

The first step is tracheotomy and the introduction of Hahn's aseptic compressed sponge-canula into the trachea. The larynx is then exposed, but is not opened until the canula has been in position at least ten minutes, in order to give the sponge full time to expand and occlude the trachea hermetically. After the larynx has been divided, its two halves are carefully held asunder with retractors or with two strands of strong silk passed

through the anterior portions of the wings, and a comparatively large aseptic sponge secured by a long string is introduced through the larynx into the lower part of the pharynx, so as to protect the field of operation from mucus and saliva.

The whole of the side of the larynx to be operated upon is then mopped twice with a 5 per centum solution of cocaine, to contract the capillaries on that side and restrain parenchymatous bleeding. Arrangements should have been made for thorough illumination by aid of the frontal mirror or of an electric lamp.

The parts to be removed are then circumscribed as far as possible between two semicircular or elliptic cuts uniting in front and behind through the whole of the soft tissues and down to the perichondrium, and at a distance of from half an inch to an inch from the periphery of the growth, so as to insure section in the healthy tissues. The growth is then to be held in dressing-forceps, and the whole area circumscribed is to be cut out with curved scissors, the points of which are to be firmly pressed against the inner aspect of the cartilage. The base should be firmly scraped with a sharp spoon, the bleeding be carefully stopped, even by ligating small vessels if requisite, and the whole interior of the wound be dusted with iodoform or with iodoform and boric acid mixed in equal parts, and the sponge canula be immediately removed. The whole of the wound is then covered with cyanide gauze.

Morbid Growths of the Larynx—Diffuse Subglottic Myxoma Radically Removed by Direct Access through an Incision into the Trachea.

This case is reported by Dr. Charles H. Knight, of New York (*New York Medical Journal*, 1894, No. 835). The patient was a woman, forty-eight years of age. The growth was found to involve the entire circumference of the windpipe, from the under surface of the vocal bands to a point one-half to three-quarters of an inch below the lower limit of the tracheal incision. This incision was two and a half inches in length, made over the upper rings of the trachea. The operation was easy and the results excellent. A photomicrographic illustration of a section of the growth accompanies the report.

The case is unique in the character and location of the growth and in the operation performed for its extirpation.

Fracture of the Larynx.

In *The Medical News* (1895, No. 1154) Dr. Thomas J. Harris, of New York, contributes an article on "Fracture of the Larynx," with the report of a case. A summary of the subject is given, with the details of twenty-nine cases reported since Durham's record of sixty-two cases in 1881.

Dr. Harris's case occurred in a member of the United States Band at West Point, who, sometime previous to the accident, had been compelled to play eleven different musical instruments. While playing the baritone, three years before, he had suddenly felt a pain in the right side of the neck, which

was accompanied by great swelling, extending to the right ear; loss of voice, dysphagia, and dyspnœa. Subsequently there was occasional discharge from both ears, with great deafness. Persistent headache set in two months later, and had been distressing. Cough had been persistent. Swelling continued for over a year, and subsided gradually.

The fingers pushed under the right ala at its upper border could detect a distinct crepitus when the patient swallowed, while he stated that he felt a marked rubbing of the two bones in the act. Pain was also produced when the cornu of the hyoid bone on the right side was grasped. Examination of the larynx with the mirror revealed nothing beyond signs of a chronic inflammation, although the false bands have always been rather acutely swollen.

Although repeated examinations of the patient have been made, Dr. Harris has never been certain how extensive was the injury. He thought the most probable condition was one of fracture of the superior cornu of the thyroid on the right side, with loss of function of the right lateral thyro-hyoid ligament. Whether there was any associated injury of the cornu of the hyoid was scarcely possible to determine definitely.

Electrolysis in Spurs and Deflections of the Septum Narium.

Dr. Karl Hess (*Münchener med. Wochenschrift*, 1894, No. 39), in an article on this subject, expresses his preferences for the bipolar method with platino-iridium needles, kept in action from three to five minutes during the earlier sittings, and six to ten minutes in the later ones; the current being increased gradually and then gradually decreased during the sitting. Eight to ten days intervene between the sittings, and four to six are necessary, so that the treatment requires five to seven weeks.

OBSTETRICS.

UNDER THE CHARGE OF

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Rupture of the Uterus in Labor Obstructed by Dislocated Kidney.

A rare and interesting case of rupture of the uterus is reported by Albers Schönberg (*Centralblatt für Gynäkologie*, 1894, No. 48). The patient had already borne two children, both in difficult labors, the first child being

stillborn. During her third labor, pain suddenly ceased, with symptoms of collapse and vomiting. A dead child was delivered by version and extraction, the placenta removed, the rupture in the anterior wall of the uterus recognized, and the patient immediately transferred to a hospital. As there was no bleeding, and the uterus was well contracted, the abdomen was not immediately opened. On the fifth day after admission, and upon symptoms of intestinal occlusion, cœliotomy was performed. The bowel was found paralytic, but no other abnormality was discovered. The patient survived but a few hours.

On post-mortem examination the left kidney was found in the hollow of the sacrum, its upper extremity in front of the promontory. The renal artery took its origin from the bifurcation of the aorta, and the renal vein emptied into the vena cava. The hilus of the kidney was directed toward the right. It was possible for a child to present by the head normally and for the kidney to offer but little obstruction to its birth. When, however, the child presented in the second position, the kidney afforded an obstacle 2 cms. thick to the passage of the child. The pelvis was a simple flat pelvis, with a true conjugate of 9½ cms. It was remarkable that so little bleeding and such slight shock followed the rupture of the uterus.

Indications for the Induction of Labor.

In a discussion upon this topic before the British Medical Association Barnes drew attention, among other causes, to excessive nervous tension as indicating a pathological condition requiring interference. Chorea and epilepsy are extreme types of this disorder, as are mania and marked emotional disorders. Excessive vomiting, especially in the latter months of gestation, is also considered an indication.—*British Medical Journal*, 1894, No. 1773.

Symphysiotomy for Symmetrically Contracted Pelvis.

Engstroem, of Helsingfors, reports in the *Annales de Gynécologie*, December, 1894, a symphysiotomy in a patient, aged thirty-one years, who had lost her first child after a difficult labor in breech presentation. Her pelvis was symmetrically contracted, the true conjugate being a little less than 8 cms. The patient was allowed to go to term, when a living child, whose head measured 9 cms. in the biparietal diameter, was delivered after symphysiotomy. The mother was convalescent and able to walk in three weeks after the operation. The usual incision was practised, the wound entirely closed and hermetically sealed, and the patient's pelvis tightly bandaged with a binder and rubber adhesive straps. The head of the child showed a slight depression where labor-pains had driven it against the promontory of the sacrum.

Wheeler (*Boston Medical and Surgical Journal*, 1894, vol. cxxxi., No. 26), performed symphysiotomy upon a multipara who had had difficult labors; in each instance in which the child had been of average size it had been stillborn. The endeavor was made to bring the head to engage by pressure and by the use of the Tarnier forceps for fifteen minutes. Symphysiotomy was then performed in the usual manner with the Galbiati knife. The child was

delivered with forceps, the cord being tightly drawn about the neck. It was cut, and the child resuscitated. During the passage of the head the symphysis separated two and a quarter inches. The true conjugate of the pelvis was 8 cms.; the biparietal diameter of the child's head was $9\frac{8}{10}$ cms. The pelvis was immobilized with adhesive plaster, and the wound closed without drainage. During the puerperium the patient suffered slight phlebitis in the left leg, which is ascribed by the writer to keeping her lying too long upon her back, namely, three weeks. At the end of the fourth week, by examining the patient while standing and swaying from one foot to the other, motion of perhaps a third of an inch was felt in the symphysis.

Puerperal Neuritis.

In the *Deutsche med. Wochenschr.*, 1894, No. 50, Bernhardt reports three cases of puerperal neuritis; in one, the median and ulnar nerves of the right side were affected; in the second, the left perineal nerve; and in the third, the right median and ulnar nerve. In the second case the forceps was applied, the head of the child remaining high in the pelvis in the second position before the use of the instrument. In the third case, the patient was ill for four months after labor, suffering with a bloody discharge and fever. All the cases presented the typical signs of neuritis, and were but little affected by treatment.

Rupture of the Uterus During Version.

In the *St. Petersburger med. Wochsenchr.*, 1894, No. 47, Armin Treu reports the case of a multigravida whose child died within the womb, and in whom labor ceased by reason of absorption of putrid material from the child. On examination, the lower portion of the uterus was found bound down by an old and firm adhesion extending across the posterior surface of the lower uterine segment. In performing version, the lower uterine segment ruptured just above this adhesion, the rupture being complete. The fœtus and placenta were, however, removed manually, and the patient made a complete recovery.

Treatment of Brow and Face Presentations by Manual Interference.

In the *Zeitschrift für Geburtshülfe und Gynäkologie*, 1894, Band xxxi, Heft 1, Thorn contributes a paper on the "Treatment of Brow and Face Presentations by Manual Interference." He reviews the literature of the subject, and, from his own experience and that of others, believes that when a face presentation develops at the end of the first or second period of labor, that delay is dangerous in the interests of the child. The first effort of the obstetrician should be to change the presentation into an occipital one by combined manipulation under anæsthesia. For the success of this manœuvre, it is requisite that the fœtus be movable in the pelvis, and that the mouth of the womb be sufficiently open to permit the introduction of at least half a hand. The combined method is contra-indicated when the face presentation was present from the beginning and did not develop through abnormality in the

mechanism of labor; also contra-indicated in prolapse of the umbilical cord or of the fœtal extremities in placenta prævia and low attachment of the placenta, where the lower uterine segment is greatly distended, and where disproportion between the fœtus and pelvis exists.

In cases where the pelvis is normal, if the combined manipulation fails, and there is no immediate danger to the child, it is best to wait to observe whether the attempt to alter the presentation may not result in increased labor-pains and further progress in the labor; when, however, this improvement does not occur, internal version should be performed. Where the pelvis is contracted version and extraction by the feet should follow.

In brow presentations, when the brow is found above the brim of the pelvis, and movable in the pelvic inlet, the pelvis being normal, the attempt should be made to convert the presentation into one of the vertex by manipulation. When this fails, unless labor proceeds spontaneously, internal version must be performed. If brow presentation occurs as a complication of contracted pelvis, even in slight degree, delay should not take place, but failure in combined manipulation should lead to a prompt resort to version.

The Treatment of Complete Rupture of the Uterus.

Cholmogoroff (*Zeitschrift für Geburtshülfe und Gynäkologie*, Band xxxi., Heft 1) reports the following interesting case. The patient was a multipara who had borne four children by difficult labor, the last of which was terminated by craniotomy, with laceration of the anterior wall of the neck of the uterus. The patient recovered completely in three weeks. Examination of her pelvis showed a justo-minor pelvis of moderate contraction. The fifth pregnancy went on normally. When labor commenced, hemorrhage of moderate degree ensued. The patient was under the care of a midwife, who ruptured the membranes. The labor, however, failed to proceed, and the patient was brought into the hospital. On admission, examination showed the fœtus with its long axis parallel with that of the uterus. The head could be felt above the usual position. No heart-sounds were present. On vaginal examination the mouth of the uterus was found fully dilated. In front and to the right could be felt a considerable portion of the placenta. The breech of the child was at the pelvic inlet, the sacrum directed posteriorly and to the left. The right foot was found on the same level with the breech. Hemorrhage, although not excessive, persisted. Accordingly, the right foot of the fœtus was grasped; following this, the hemorrhage did not abate, and accordingly extraction was immediately carried out. As the breech was an inefficient tampon, the hemorrhage continued. The extraction of the child was a most difficult one, and recourse was finally had to craniotomy upon the after-coming head, the opening of the skull being made through the palate. After the extraction of the child, hemorrhage did not entirely cease, and an examination showed the transverse rupture of the uterus at the cervix. The placenta lay in the abdominal cavity among the intestine. The fœtus also had been in the abdominal cavity. After the removal of the fœtus and placenta the hemorrhage did not cease. The condition called for a choice between two methods of treatment, one by cœliotomy, the other by the use of the tampon. As the fœtus had been in the abdominal cavity for some little time, and the

entire labor had not been conducted antiseptically, infection of the peritoneum was probable; this would indicate treatment by the tampon. On the other hand, the persistent hemorrhage called for suture of the ruptured uterus. With the idea of accomplishing all that could be done by both, the rent in the uterus was stitched *per vaginam* through the greater portion of its extent. A long glass tube was passed through the rent into the abdominal cavity, and the vicinity of the rent irrigated with two per cent. solution of boracic acid. A Sims speculum was introduced, the borders of the rent seized with tenaculum forceps, and the greater part of the rent closed by suture. At the left angle of the rent an opening sufficient to admit two fingers was allowed to remain. The bleeding immediately stopped. Through this opening iodoform gauze was passed into the abdominal cavity, and the end allowed to remain in the vagina. The uterine cavity and that of the cervix were tamponed with iodoform gauze; this was allowed to remain for four days, when it was removed and the vagina tamponed with fresh gauze. In eight days the tampon was removed from the abdominal cavity. The patient suffered from mastitis, but otherwise made a good recovery. Cholmogoroff draws attention to this combined method of treating complete rupture of the uterus as combining the advantages of both cœliotomy and the use of the tampon.

The Induction of Labor in Contracted Pelves.

Beuttner (*Archiv für Gynäkologie*, 1895, Band xlviii., Heft 2) reviews thoroughly the modern literature of the subject, and adds an account of thirty cases in which labor was induced because of contracted pelves. His conclusions from his study of this procedure are decidedly in favor of the induction of labor when compared with other operative procedures. He would place primigravidæ and multigravidæ in the same category, regarding the history of a previous difficult labor in multigravidæ as of very little importance. He considers 10 cms. not too large a true conjugate to justify the induction of labor. The element of importance in deciding upon the operation is a comparatively slight disproportion between the size of the head and that of the pelvis; to determine this, he is convinced of the value of Müller's procedure, and would induce labor when the head of the child can be brought to engage in the brim of the pelvis only by long and vigorous pressure exerted externally and from above; to determine this fact accurately anæsthesia is often necessary. The duration of pregnancy and the supposed development of the fœtus at a given period are unreliable factors upon which to base an operative procedure.

Two Cases of Spontaneous Rupture of the Uterus.

In the *Centralblatt für Gynäkologie*, 1895, No. 2, Gessner reports two cases of spontaneous rupture of the uterus. The first occurred in a multipara who had had easy labors. In the present instance a transverse presentation developed, with prolapse of the arm. While the attending physician was preparing to do version, the cord, which had prolapsed, suddenly ceased to beat; uterine contractions stopped, although hemorrhage was absent. The child was extracted and easily resuscitated, when it was found that a complete

rupture of the uterus had occurred. The patient was at once transferred to the clinic of the University of Berlin, where cœliotomy was performed by Olshausen. Suture of the uterus and drainage by gauze tampon were employed. The patient made a good recovery.

Gessner's second case was that of a patient whose labor was delayed by hydrocephalus. The attendant physician had applied forceps, but failing to lock them, had desisted from making traction. Rupture of the uterus occurred, and the patient was transferred to the clinic, where the fœtus was easily extracted after craniotomy. In this instance it is doubtful whether the rupture was complete. It was treated by gauze drainage without cœliotomy. The patient made a good recovery.

In the first instance it is interesting to note that in transverse presentations as pointed out by Freund, it is not the uterine muscle which ruptures, but the attachment of the vagina to the uterus which is drawn away. The prolapse of the arm seems to be an important factor in such an accident. In the second case the rupture was typical of the variety described by Bandl, occurring at the junction of the upper with the lower uterine segment.

PÆDIATRICS.

UNDER THE CHARGE OF

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THE USE OF HIGH INTESTINAL INJECTIONS IN CHILDREN.

SSOKOLOW, of St. Petersburg (*Jahrbuch f. Kinderheilkunde*, Bd. xxxviii. S. 186), has made an extensive anatomical study of the intestines of children to determine to what extent the ileo-cæcal valve offers an obstacle to the passage of an injected fluid from the cæcum to the ileum. After noting the different positions of the rectum and the sigmoid flexure, which vary so frequently in children from the usual positions as found in the adult, he finds that the distance to which the tube is inserted does not materially affect the passage of fluid to the whole extent of the large bowel, provided a sufficient pressure be obtained. It is desirable, however, to reach the sigmoid flexure, in order to avoid over-distention of the rectum and consequent forcible expulsive efforts. The dorsal position, with thighs drawn up, and pelvis elevated, is found to be the most advantageous, and a pressure of not more than 1 to 1½ metres is advised.

Among 200 subjects experimented upon, 130 being under one year, it was found that the ileo-cæcal valve was competent in only 27 cases (20.8 per cent.), while in 103 (79.2 per cent.) water passed freely through the valve into the small intestine. In older children, from one to twelve years, insufficiency

was not so common, since out of 70 cases 37 had incompetency. A pressure of more than 3 metres was dangerous, being liable to rupture the gut or disorganize the valve. It is, therefore, evident from this study that in children under one year fluid may be expected to reach the small intestine in three-fourths, and in older children in one-half of the cases. Therefore, in every suitable case an effort should be made to reach the small intestine with the irrigating fluid. Besides the numerous morbid conditions admitting of this treatment (dyspepsia, koprostasis, entero-colitis, cholera infantum, dysentery, etc.), the possibility of intestinal feeding, as Muselli has suggested, must not be overlooked. This is especially valuable in such conditions as trismus, laryngeal diseases or palsy of laryngeal muscles, dysphagia from stenosis of œsophagus, and diseases of the stomach where rest of the organ is demanded.

The Digestibility of Sterilized and Unsterilized Milk.

Bendix (*Jahrbuch f. Kinderheilkunde*, Bd. xxxviii. S. 393) has made the following interesting conclusions based upon experiments in feeding three children (aged one and three-quarters, two, and two and a half years) for equal periods (four days, six days, and seven days respectively) upon two dietaries differing only in the employment of Pasteurized or sterilized milk, the most rigorous estimation of ingesta and excreta being made in each case.

With a child in good health there is no difference in the assimilation of nitrogen or fat in favor of Pasteurized over sterilized milk, whence it follows that sterilized milk is as well digested and as completely absorbed as non-sterilized milk. But with a child whose digestion is weakened by diminished secretion of bile, assimilation is diminished, as compared with the normal, in the proportion of 0.9 to 2 per cent. for nitrogen, and 2.4 to 3.8 per cent. for fat; but it is diminished equally for the two varieties of milk. The taste and odor of milk remain good after sterilization, though differing decidedly from that of raw milk; but it is taken as readily as Pasteurized milk.

Contrary to the opinion of Kalischer, the author has never found that the brownish color of sterilized milk is liable to cause young children to refuse it. He has never seen sterilized milk produce the slightest disturbance of digestion; on the contrary, the general condition and appetite have been good, the stools normal, growth regular, and vomiting never occurred.

On the other hand, since sterilization prevents every danger of transmitting grave diseases by destruction of pathogenic microbes, while it prevents other diseases, especially of the intestines, by destroying the germs of fermentation and decomposition, the physician should insist upon its use in preparing milk for the artificial rearing of infants. Sterilization is preferable to Pasteurization, since the latter does not insure so completely the destruction of bacteria, especially the spores.

A Floating Kidney in an Infant.

In the course of a paper on floating kidney William W. Stewart (*Medical Record*, February 9, 1895, p. 163) reports a case accidentally encountered in an infant of eight months. The child had swallowed some pieces of plaster,

and intestinal obstruction followed. After thirty-six hours of ineffectual treatment the author was called upon to operate. Though the abdomen was quite tympanitic, a distinct movable tumor could be felt on the right side halfway between the crest of the ilium and the umbilicus. Its exact consistency and shape could not be made out. This tumor was believed to be the obstructed gut. The mother was questioned carefully, but stated she had never before noticed the tumor, which she could now feel with the greatest ease. Upon opening the abdomen the tumor was found to be a floating kidney, while the obstruction was located in an intussusception in the left hypogastric region. The child had never suffered any inconvenience from the kidney. The author believes this to be the youngest case of floating kidney yet reported.

Trional as a Hypnotic for Children.

Claus (*Internationale klinische Rundschau*, 1894, No. 45, p. 1613) makes a report of his experience with trional as a hypnotic for children. Given in doses of three grains to twenty two and one-half grains, according to the age of the child, he has found this drug a reliable hypnotic, producing a physiological sleep and leaving no headache or heaviness on the following morning. The patient does not become accustomed to its effects. Sleep is usually produced ten to fifteen minutes after ingestion. In painful conditions its influence is not very satisfactory. Upon the intellectual, respiratory, and circulatory functions its action is negative. One instance, however, is mentioned in which, in a case of broncho-pneumonia in a child of five years, a single dose of eleven grains produced ataxic symptoms. The most satisfactory results were obtained in cases of chorea and pavor nocturnus, and in insomnia from disturbances of dentition or of digestion. In several cases of the latter class the influence of the drug upon the digestive functions seemed beneficial. This agrees with previous experience in the use of trional among the insane. The dose from one month to one year is three to six grains; from one to two years, six to twelve grains; from two to six years, twelve to eighteen grains; and from six to ten years, eighteen to twenty-two and one-half grains. It is best given half an hour after the evening meal, or fifteen minutes before bedtime. It can be given in hot milk, or in confection or honey.

Bursal Enlargements as Indications of Incipient Tuberculous Arthritis.

D'Arcy Power (*British Medical Journal*, 1894, ii. p. 412) calls attention to the importance of bursal enlargements in the neighborhood of the hip-joint in children. Such cases at the time show no evidence of affection of the joint. There is no limp, no pain, no muscular fixation, and the head of the bone moves freely in the acetabulum. Yet such bursal enlargements are of very serious import and are of great diagnostic value, for they frequently are the first indications of tuberculous mischief occurring in a patient who otherwise appears sound. The tuberculous trouble thus starting in the bursa will spread rapidly to the joint, unless care be taken to prevent it. In support of this six cases are reported, taken from the records of the Victoria

Hospital for Children during the current year. If so many cases have come under the observation of a single surgeon in a comparatively short time, the author believes they cannot be of infrequent occurrence, but very little reference to the condition can be found in most text-books. If the effusion into the bursa be seen before suppuration has occurred, the fluid should be evacuated by incision rather than by aspiration. If suppuration has occurred, the abscess must be opened, scraped, thoroughly cleansed, and every effort made to get it to heal at once. Suitable precautions are to be taken at the same time to secure physiological rest for the joint, while the general hygiene of the patient must be improved, for it must not be forgotten that the patient has already passed the threshold of tubercular disease. The drainage-tube should be dispensed with, in order to obtain union by first intention.

PATHOLOGY AND BACTERIOLOGY.

UNDER THE CHARGE OF

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Osteomyelitis.

Closely associated with the problems of suppuration are those connected with the etiology of acute osteomyelitis. It, too, was at first supposed to be a specific disease of the bone-marrow, and Becker consequently designated the micrococcus which he discovered in the pus the "osteomyelitis coccus." A very careful article, from the pen of Max Jordan (*Beiträge z. kl. Chirurg.*, 1893, x. 585), reviews the whole subject from the bacteriological point of view, and adds to the cases of osteomyelitis previously reported nineteen of his own, in all of which bacteriological examinations were made.

Histologically acute osteomyelitis is usually a purulent inflammation, occasionally it is of a serous or of a hemorrhagic-septic type. Notwithstanding this the identity of these forms is shown by their identical etiology and similar clinical type. It is almost always a secondary process, though the primary focus of inflammation may have disappeared before the onset of symptoms of osteomyelitis. In these cases the osteomyelitis is frequently mistaken for the primary disease. Its onset is not infrequently very insidious. It usually affects first the ends of the long bones at or near the line of junction of epiphysis and diaphysis, and is almost exclusively a disease of the period of bone development, its rare occurrence in adults being explained by the fact that in them the special conditions predisposing to the disease during the growing stage of the bone have ceased to exist.

As a number of quite distinct species of bacteria have been found associated with osteomyelitis it is evident that Becker was wrong in supposing it to be a specific disease. It is most often caused by the *staphylococci*, aureus, albus,

or both, but may be caused by the *streptococcus pyogenes*, by the *pneumococcus* of Fränkel, or by the *bacillus typhosus*. It has been produced experimentally in animals by the soluble products of these bacteria and also by purely chemical irritants. The paths of entry of these germs into the body are the same as in other infectious processes—through the alimentary tract, the respiratory tract, or the skin. A number of interesting cases are given by Jordan in which osteomyelitis occurred secondarily to boils, whitlows, and other comparatively insignificant lesions of the skin which ordinarily attract but little attention. In many of these cases cultures showed the same species of bacteria in the original skin lesion and in the osteomyelitic pus.

As far as etiology is concerned, then, acute osteomyelitis stands in the same relation to its cause that suppurative inflammation in other tissues occupies to the same causes acting on them. Since it is almost invariably secondary and is often associated with a number of other suppurative lesions in widely separated parts of the body, Jordan considers it just to regard it as part of a purely bacterial form of pyæmia which shows a great disposition during youth to the formation of metastases in the bones because of a peculiar predisposition afforded by them during the developmental period.

Mixed Infection in Pulmonary Tuberculosis.

Notwithstanding the proof of Fränkel and Troje (American Journal of the Medical Sciences, June 1894, p. 744) that the tubercle bacillus alone is quite competent under proper conditions to produce pulmonary tuberculosis of the most diffuse nature, the impression has steadily grown that in very many cases of phthisis a "mixed infection" occurs at some time in the course of the disease, and that an important *rôle* in determining the progress of the disease is played by the bacteria causing the associated lesions at such times. This impression has had its foundation in the frequent clinical observation of exacerbation of chronic phthisis during and after influenza, lobar pneumonia, and after the various complicating broncho-pneumonias, as, for example, after the broncho-pneumonia of measles in children. It has been suggested that were it not for the contributing conditions of the mixed infection the body would be able to protect itself in a very large proportion of cases from encroachment of the tubercular process. Whether this latter extreme view be correct or not, it becomes of the greatest importance from both prophylactic and therapeutic standpoints to determine the germs most frequently contributing to the progress of the tubercular process, and to investigate the most efficient means of lessening their influence.

With a view to throwing light upon the first of these questions, Spengler, working at the Institut für Infectionskrankheiten, in Berlin, under Koch's guidance, has examined the sputum of fifty patients suffering from phthisis, using every precaution to exclude any sources of error. (*Zeitschr. f. Hygiene u. Infectionskrankheiten*, 1894, xviii. 343.) Stained cover-glass preparations of the sputum and cultures from it by both Pfeiffer's and Kitasato's methods were carefully studied, and careful microscopic and bacteriologic examinations of the diseased organs were made in all the fatal cases. In only five of the fifty cases studied was the tubercle bacillus alone found. Three of these died, and in the lesions of all the tubercle bacillus was alone.

In nearly eighty per cent. (39) of the cases the *streptococcus pyogenes* was associated with the tubercle bacillus, and in twenty-one of these the streptococcus was believed to have been an active agent in the production of the lesions, because of its presence in large numbers in the sputum coincidently with the occurrence of fever of a peculiar type, and because of its presence in the lesions post-mortem. In fifteen cases the streptococcus appeared to be present as a passive accompaniment of the tubercle bacillus. In these there was comparatively little fever, the progress of the cases was very slow or stationary, and in the majority the streptococci seemed to be present as a residue of an acute condition which had subsided some time previously. Eight of these cases had had the advantage of a residence at Davos, in the Tyrolean Alps; and Spengler suggests that the benefit therefrom depended chiefly upon the rapid recovery from the associated affection, the streptococci in all these cases quickly diminishing in numbers, and in several of them ultimately disappearing entirely from the sputum.

The *diplococcus pneumoniæ* was found to be the associated germ in one case, and in one each the *micrococcus tetragenus*, the influenza bacillus and the pseudo-influenza bacillus were associated with the streptococcus.

The important practical deduction from this work is that in the prophylaxis and the treatment of tuberculosis of the lungs careful consideration should be given to the part played by the associated bacteria. Persons affected with slight or transitory inflammations of the respiratory tract should be removed as far as possible from all sources of infection by the tubercle bacillus; they should under no circumstances be placed in the same wards in hospitals with consumptives. Similarly, great care should be used to exclude from persons already affected with tuberculosis all sources of infection with the bacteria which have been shown to exert so deleterious an influence upon the progress of the disease. This would seem to be best accomplished by the removal of the tubercular patient to a high mountainous region, or to one at all events where the air is free from the germs of suppuration and pneumonia. And, furthermore, in the treatment of cases of phthisis, the first care of the physician should be to rid the lungs as quickly as possible of the associated bacteria, for so long as their hurtful influence continues it is useless to institute specific treatment of the tuberculosis.

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